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# NUTRITION AND DEVELOPMENT





CLINICAL PEDIATRICS

# NUTRITION AND DEVELOPMENT

BY

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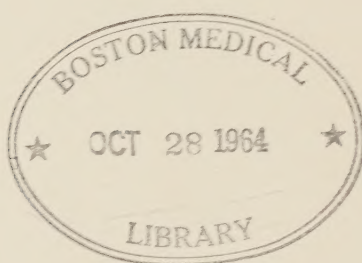
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## PREFACE

The intent of this volume is to visualize for the general practitioner in a consecutive, panoramic manner, the processes of digestion and metabolism, together with the resulting phenomena of growth and development of all stages of childhood up to puberty. The several food elements have been described, and the part played by each in the human economy explained.

The subject has been presented in a way intended to aid the physician in clearing up his problems pertaining to all phases of nutrition. Encroachment on the scope of any other volume in this series has been avoided as far as possible, and yet in order that an orderly sequence be maintained in the story of development from the fructified ovum to the period of puberty, it has been necessary to describe certain facts and details belonging to some of the other authors. Slight repetition of this sort, far from detracting, will probably serve a double purpose: added emphasis and restatement of facts from differing angles.

Chemical and biological processes have been presented in this work in such a manner as to be available to the man who is so busy as to be unable to keep adequately in touch with the rapid changes occurring in the subjects discussed.

A large amount of research has been done both in this country and abroad on the phenomena of growth and nutrition; extensive and intensive studies in metabolism and nutrition have been undertaken during the recent past and much information has been gained, but the reports of the results are scattered in the literature to such an extent that much time is required to locate even a single fact.

The present volume attempts to bring together in a readily available form the salient facts heretofore widely scattered, and thus enable the reader to gain a comprehensive picture of the physical life of the growing human organism.

To all authors whose works have been consulted, the author of this volume expresses his acknowledgment and gratitude.

To his assistant, Dr. W. W. Waddell, Jr.; to Dr. Henry B. Mulholland; to Dr. A. Chanutin; to his secretary, Miss Edith Hornsby; to

Miss Grace Brinton and Miss Verna A. McKean, dietitians; to Margaret Knowles Speidel, artist; and Arthur Jehial Weed, photographer; to Dr. R. Cannon Eley, Dr. L. R. Broome, and Dr. E. H. Cunningham, for charts and tables, and to Mr. Finlay Forbes Ferguson, who materially aided in the correction of the manuscript, the author wishes to make sincere acknowledgment and to express his appreciation.

LAWRENCE T. ROYSTER.



## PUBLISHERS' ANNOUNCEMENT

The publishers take pleasure in presenting to the medical profession the series of monographs of which this volume forms a unit.

The many inquiries which reached them proved, in advance of publication, that the work should be in monographic form and clinical in its presentation.

The series when completed will, they believe, be the most useful for the audience for whom it is written, the general practitioner of medicine, that has been presented in its particular field.

The authors are all men of wide experience and, in the main, teachers. The combination makes the work authoritative and of the utmost service in a field which has been often termed a "therapeutic specialty."





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# NUTRITION AND DEVELOPMENT

## CHAPTER I

### INFLUENCE OF FOOD ON HUMAN DEVELOPMENT

It is exceedingly difficult to appraise properly the importance of the many factors, influences, and circumstances which determine the course and results of great events. Nowhere is this more difficult than in the development of the human race. The importance of any influence is relative to that of some other influence; hence, it is quite impossible to state with any degree of certainty that any one factor is the most important in such an extensive series of events as the development of mankind. If, however, one were called on to state in a single word what influence had been the most potent in this development, it is quite safe to say that that one influence is *food*.

With the first appearance of life on this planet food at once became the most essential consideration. When the lowest form of vegetation made its first appearance, the struggle for food (entirely inorganic) commenced. Likewise, when the first unicellular form of animal life emerged, the acquiring of food, both inorganic and organic, became the supreme object of effort. The plants first absorbed their nutriment from the gases of the air, water from both air and soil, and inorganic matter from the soil. These inorganic substances were transformed into organic and so the endless circle of transmutation was begun. Animals first subsisted on this vegetation but later added animal food to their dietary by preying on each other. Both animal and vegetable individuals, however, immediately began the same cycle of birth, growth, development, decline, and death; returning to the soil to enrich the earth, their disintegrated forms to be incorporated into other plants and animals, to be consumed or otherwise utilized again; and thus was developed a perpetual reincarnation. *Omnia mutantur, nihil interit.*

Coincident with the appearance of life, therefore, there developed a twofold struggle—for food, on the one hand, and for the propaga-

tion of kind on the other; that is, the struggle for existence, or the perpetuation of life. The necessity for artificial shelter arose only when animal life developed high enough to be considered in the light of what we are pleased to call the dawn of civilization. These two aims are, therefore, the prime essentials, and once established must continue as long as life remains in any form.

The survival of life, either plant or animal, was conditioned on an ample food supply and a congenial environment. This is certainly true of plants, but to a less extent true of animal life, for the latter has shown itself adaptable to climatic environment provided the food was adequate. So the necessary search for food and the ability to find it in a certain locality have probably been the most potent factors in the development of tribes and species, which differ in many respects from the parent stock. This localization of small groups of individuals (tribes) with the consequent acclimatization, has been productive of such variations from parent stocks as to give rise to races. The characteristics of each race are so marked that it is readily distinguished from every other race. These characteristics include stature, coloring, form and feature, and many others too numerous to mention. We are apt to regard these as developments or variations due to climatic influences; the locality in which the tribe settled and the race developed. We must remember, however, that what actually determined the place of abode was the finding of an adequate food supply, and that the kind of food which grew in a particular locality probably developed distinguishing racial characteristics rather than the climate or other local influences. It is not unreasonable to suppose that species might have been brought about in the same manner.

Concrete examples of the influence of food on stature are not wanting. A race relatively short of stature may, under the influence of better food conditions, provided this influence is sufficiently prolonged, actually increase in stature. This fact is peculiarly well illustrated by the Japanese, who are notably a small race. However, those members of this race who have been born and reared for a generation in the Philippines—that is, largely under the influence of American food standards—average two inches taller than their ancestors or others of their race remaining in Japan. Several theories must be considered when we study such a remarkable fact as this. It may be argued that climatic conditions alone may have been the determining factor; it would appear to be rather that climate, if influential in this instance, was productive of a better food supply. On the other hand,

American food standards have largely replaced native standards. The reasons for this may be more complicated than at first appear. An improved food supply alone may have furnished the material for growth which is lacking in the original country, for it is well known that the diet of the Japanese is almost entirely devoid of protein. Such a diet as this, over long periods of time, could well produce a stunted race, since the necessity for protein in promoting growth and development is an established fact. On the other hand, it may be that certain food factors have been lacking which activate the cells to improved metabolism. In this connection it is well to bear in mind the fact that for the first year or more of life (certainly during the nursing period) Japanese children compare almost identically in height and weight with American children, showing that whatever food the mother may subsist on, she transmits the essential elements to her offspring, and that the stunting occurs after artificial feeding is commenced. We must also bear in mind the fact that certain foods are sufficient to maintain life, but that certain others are essential to growth and development.

Another racial example is of interest. For a definite period after the Franco-Prussian War the average height of the French people was two inches lower than it was before that war. Some have ascribed this to the fact that the tall men were chosen for the army and were killed off in large numbers, and that the population of the next generation was largely the offspring of inferior parents, at least on the male side. It is quite conceivable, however, that the food supply during the periods of pregnancy and lactation of the mothers of this and the succeeding generation, and of the children themselves after the nursing period was over, was such as to inadequately furnish material for growth. It is natural to presume that these children inherited some height factors from their mothers. It is known that during this post-war period a considerable number of women of superior stock remained celibate rather than marry inferior men. Hence, the influence of the height factor on the female side is difficult to appraise.

We may assume, then, that food has played a very important part in the development of mankind, in determining the localities in which tribes settled in the course of their wanderings in search of an ample supply of food, and there grew into races and nations. On the other hand, the failure to find an ample supply, or, at a more advanced period, the failure to find a locality whose soil and climate were capable of producing such a supply, has probably caused to perish many tribes that

were unable to battle with the obstacles. Some of these may have been undesirable stock and their disappearance from the face of the earth no loss.

But it is not alone in the development of mankind and the determination of races through location that food has been of importance. In the not very distant past, nation has fought nation for the possession of food when the supply was scarce with the one and abundant with the other. As a result of this, still other nations, or at least tribes—there is merely a relative difference—have been made to perish, either as the result of the combat or the famine which followed. The sudden failure of food supply due to natural phenomena has often resulted in similar catastrophes. In more recent times, the outcome of campaigns and the final result of wars have been often determined by the food supply more definitely than the supply of munitions. It is a well-established fact that the final success of the Allies during the World War was due as much to American wheat as to American men and arms. And not only has the quantity of food been the determining factor in such instances, but even where the supply was ample the quality has been deficient, for scurvy has in a number of instances so debilitated an army that it was unable to fight. Thus, the lack of a single food factor has turned the tide against an otherwise strong and efficient army. It has been said the cause of failure of the Mesopotamian campaign during the late war was due to this deficiency disease. This last statement brings us to a consideration of the relation of the quality of the food supply rather than its quantity, not only in the past and during the developmental period which we have been considering, but, what is of infinitely greater importance, to us at the present time.

Are we a well-nourished or a mal-nourished people? Are we better or more poorly nourished than the people of the recent or remote past? Do the children of to-day weigh more or less for their height than did those of former generations and remoter periods of history? There has certainly been in the immediate past a large amount of nutritional disorder, and until quite recently—that is, before the newer knowledge of nutrition was possessed and applied—deficiency diseases were very prevalent. Standards of comparison are wanting by which we might answer some of these questions and much must be inferred from what we are able to deduce from historical records and by reading between the lines of these same records. One fact stands out with striking prominence, however, and that is, that the children of the past



twenty-five years weighed more for their height than did those of the closing years of the last century, if we may judge the latter by such records, rather inaccurately kept it is true, which are available for study. From this we may infer that our increased knowledge of food values and the chemistry of digestion has already begun to be effectively applied and that a sturdier race is in the making.

It is quite pertinent to ask why the people of former generations and more remote periods of civilization did not suffer from nutritional disturbances. The answer is paradoxical: they did and they didn't. Some of these conditions have been coexistent with history, some have developed in the course of and as a result of the progress of civilization. When our domestic animals ran wild and roamed the plains they were compelled to forage for their own food, and this they did with a selective instinct and chose what was good for them and produced the best milk for their offspring, and with almost unerring accuracy rejected what was useless or positively harmful, in open seasons. During the rigorous seasons, however, they were often compelled to eat whatever could be found, and as a result many fell victims to a general state of malnutrition or died. Likewise as they became domesticated as the servants of man, their food was selected for them. Instead of the plains and fertile fields, they were restricted to the narrow confines of enclosed pastures and compelled to subsist on whatever vegetation could grow there and, when this failed, on dry provender which deteriorated from drying and aging.

So it has been with man. We see him during his savage and semi-barbaric existence, feeding on fresh vegetation in its natural state, and on animal food which was consumed almost as soon as it was killed; and having no fixed habitation, he moved when food became scarce in one locality to a new home where food could be secured with ease again, finally settling where a permanent food supply existed or where a soil was found which could be induced to furnish food for himself and his domesticated animals. And when a step upward in the scale of civilization was taken and man began to use grain in powdered form as bread, it was milled in a mortar just before cooking.

So as civilization advanced through a slow and steady development, the food question changed but little until man began to live in cities. Till then, he was troubled little by the problems of nutrition except during times of famine, drought, or war, when the food supply was all but cut off, and invariably under these conditions scurvy developed. Even in the early stages of urban life proximity to the country districts

## 6 INFLUENCE OF FOOD ON HUMAN DEVELOPMENT

made it easy to obtain the necessary provender within a short time of its production. It was in about this stage of civilization economically, though not socially, that the early settlers in this country found themselves, and so they lived until density of population favored a change which was far more abrupt than any which the world has ever known.

The period of comparative simplicity of living has occupied the greater proportion of civilized life on the American continent. Meat was secured by hunting or, later, by the slaughter of domesticated animals as the need arose, and consumed while fresh because of lack of facilities for keeping. What small amount was kept was preserved by a process of smoking which retained the nutrient qualities that would have been destroyed by aging. Vegetables were consumed within a short time from gathering; dairy products were utilized almost as fast as they could be produced and prepared for consumption; while grain was harvested and garnered in comparatively small quantities and milled within walking distance of its source and consumed as soon as milled.

But gradually and inevitably increase in population, change from rural to city life, and rapidly developing economic exigencies wrought their changes. The vast majority of heads of families ceased to produce their food and depended on the purchase of needed commodities from the small farmer, who, as he rose in the scale of production, grew crops of limited variety, eventually restricting his efforts to the output of a single article. The farm receded farther and farther from the center of consumption, thereby causing delay in transit and the necessity for storage with its consequent aging. Grains of every kind, instead of being produced, milled and consumed within a radius of a mile or so, are now sent several thousand miles and returned to be consumed after months of delay, thus impairing the value of the fresh product.

With civilization also came a departure from the simple life, with its simple tastes, and certain refinements of diet were demanded by the upper classes, and this was quickly imitated by the humbler classes, so that white flour bread, almost devoid of anything but the starchy content, was substituted for the whole wheat bread, or seconds, of an earlier period.

During this earlier period were developed also certain methods of preparation, made necessary by the limited variety of food products, which formed rational, balanced diets of almost scientific precision, such as the once famous, but now often spurned, corn pone and "pot

licker," the latter being little less than a concentrated essence of vitamins.

These changes in the handling and preparation of food products, with the consequent diminution of food values, have taken place with such rapidity that the human economy has not had sufficient time to adjust itself to the change—for a century or two is a very short time in the development of a race—with the result that deficiency diseases have become exceedingly common.

An accurate knowledge of the chemistry of food has been slow in arriving when compared with the science of diagnosis and the marvels of modern surgery, but when the knowledge of food values, the chemical composition of the various elements of which it is composed, and processes of metabolism within the human body was acquired even to a limited extent, this branch of medicine or economics, by whichever name one chooses to call it, made such rapid strides that now we can safely describe it as "the science of nutrition."

Many diseases or conditions which were formerly considered infectious, or inherited, or about which nothing was known, have been shown in the light of this newer knowledge to depend for their development on the continued use of food which is insufficient in quantity or, what is more often the case and still more important, in quality. In some instances there is the lack of a single one of those subtle and as yet unanalyzed "factors" which we call vitamins; or all or any of these conditions may be fulfilled and yet there has not been sufficient contact with sunshine, for it requires the influence of sunlight for the human body to metabolize some of the food elements.

When we observe infants distorted by rickets, relieved of their deformity by the use of cod-liver oil or by exposure to sunshine, or witness the cure of xerophthalmia, through the administration of a small amount of cod-liver oil, we are impelled to marvel at the potency of food factors. When we see the symptom-complex known as pellagra, with its eruption, diarrhea, and mental disturbance, changed by a well-balanced ration from a state of apparent hopelessness to one of comfort and happiness, or a pitiful and helpless neuritic of the beriberi type restored through the administration of the polishings of rice, to usefulness and contentment, it would almost appear that the ancient art of necromancy had returned. And when we see an infant swollen, tender and racked with the pain of scurvy, relieved by the juice of a few oranges, we are almost convinced that the day of miracles has

not passed. Do not such wonders as these justify the term "the wizardry of nutrition"?

It would appear, then, that by applying the knowledge of food already possessed, we had reached a veritable metabolic utopia. This is, however, very far from being true. What has been learned during the recent past on this subject is exceedingly small in comparison with what is yet to be learned before we reach such a state. A total of four vitamins is now definitely known, and as yet each of these has been studied in relation to a very small number of conditions. Each may have an influence, probably all have, on metabolism in many other conditions, and it is almost certain that numerous other vitamins exist, concerning whose nature we as yet know nothing. The processes of digestion of the three cardinal food elements, fats, proteins, and carbohydrates, have been fairly well understood for a number of years, but as yet the rôle played by the earthy salts is only beginning to be understood and only a few have been studied to any extent at all.

It is entirely likely that those mysterious organs known as the ductless glands, and which when grouped form the so-called endocrine system, about which so much has been written and so comparatively little is yet known, play a part, perhaps an important one, in the processes of metabolism. The influence on metabolism of two of these has been partially determined; the active principle of the thyroid gland and insulin have been studied to an extent which permits practical application. It is supposed that one or more of these glands of internal secretion determine the size and shape of one or another of the body tissues, which implies that they direct the ability of this or that tissue to assimilate, or its failure to assimilate, ingested food. It is also supposed that each of these glands either stimulates or retards the activity of another of the group. Who knows but that some chemical combination of inorganic material or some unknown vitamin contained in the food we consume either activates or inactivates these glands, and so indirectly governs our growth and development?

The field of biology, especially that part of it which relates to inheritable traits and the factors which determine them, has been studied to such a small extent in its relation to nutrition, that at present our knowledge is not sufficient for practical application. The factors which determine stature, physique, muscular activity (hyper- or hypo-kineti<sup>c</sup>) are inherent, and can be controlled only by inbreeding on the one hand and crossbreeding on the other. Yet the activity of all cells and their contained factors are obviously dependent on the soil in which they



develop, the food which they assimilate. Therefore, who can tell at the present time to what extent all of these matters are influenced, indeed even controlled, by the quantity, quality, and proportion of food products with which the growing organism is supplied?

It is not the past, nor even the present to which the greatest interest attaches in the problem of nutrition. The inferences drawn from our meager knowledge of the development of mankind, through mute testimony dug from the depths of the earth in the shape of fossil remains, the evidence drawn from recorded history and the often eloquent deductions from what we visualize between the written lines, the facts determined by modern scientific experiment go for naught if we do nothing with them. Unless these experiences can be utilized for the betterment of mankind and the building of a sturdier and more stable citizenry in the future, they are worthless. Since at present we cannot control the production of the cells making up the human organism, we must do the next most important thing: supply these cells with the best nutrition possible. Thus, we become the molders, in large measure, of the social fabric of the next generation, and the next; the makers of history through the better nourished bodies of its citizens, doing our share in the advancement of civilization, and writing "the romance of nutrition."

## CHAPTER II

### FORMS AND SOURCES OF FOOD

The study of nutrition is by far the most important part of pediatrics. It is, in fact, the very foundation of this branch of medicine. It is important from the standpoint of therapeutics, for through food alone can we correct digestive disorders, and prescribe the diet, both in quantity and quality, best suited to the needs of the body while suffering from the various infectious processes. It is important from the standpoint of prophylaxis, a knowledge of the chemical composition of the food elements, their caloric value, and the processes of digestion being necessary to prevent digestive disturbances. By maintaining body weight and promoting metabolic equilibrium, through a balanced ration, we may also prevent malnutrition—the greatest foe of the growing child.

A thorough working knowledge of the processes of metabolism is essential in order for us to meet the various needs of the growing human organism. These needs are: the supplying of a food adequate to care for destructive processes taking place as a result of the liberation of energy, the supplying of an additional amount for the storage of energy and for further meeting the requirements of growth and development.

The term “food” is used to designate substances or materials which promote the usual body functions, such as: the promotion or stimulation of growth, the development of vital organs and the stimulation of the functions of these organs, the maintenance of heat and the repair of waste. This implies the transformation of material into body tissue, the ultimate object being the integrity of the cell. The term “food,” then, as usually employed, applies to a group or collection of individual substances known as food elements, each one of these being a complex chemical compound.

### THE FOOD ELEMENTS

The food elements thus referred to are: protein, fat, carbohydrate, mineral salts, and water, with the addition of the recently discovered

“accessory food substances” known as vitamins, which must now take their place in the list of food elements.

It is necessary for a human being to be supplied with a total daily quantity of food which is sufficient to meet the caloric needs of the body; but unless the proportion of the food elements is properly balanced, there is failure of nutrition or development or both at some point. We may supply a correct amount of fat, carbohydrate and protein, and in the right proportion, and yet if the growing organism does not get a definite amount of mineral salts there is likewise inadequate food, which produces untoward effects.

Again, we may supply all of these in proper amounts and in definite proportion, and still, if the vitamins are lacking, the organism not only ceases to grow and develop, but may even perish; while without water, all food administration fails in its purpose.

The form, quantity, and proportion of all these substances required by the growing child differ markedly from the needs of the adult.

The requirements of the growing child for the various food elements are taken largely from the observations of Holt and Fales.

**Protein.**—By far the most important of the food elements, if we except for obvious reasons those elements which go to make up the skeletal structure, is protein. It is essential to life, the wear and tear of life producing considerable loss of protoplasmic protein at all ages. Protein, moreover, is essential to growth, since it contains the necessary structural units of protoplasm—the amino-acids. The adult needs a supply of protein sufficient only for repair, while in the child, not only must the need for repair be met, but to this is added a more important requirement, that of growth. There is no storage of protein as such in the human body, that portion which is not immediately built into tissue being katabolized: hence it is evident that a constant supply must be furnished daily to meet these requirements.

Ingested protein alone can sustain life for a long time, but an immense amount is needed for this purpose, and the tax of such an amount on the excretory apparatus is too great. Protein is also to a certain extent a source of energy, but less so than fats and carbohydrates. These two elements, therefore, are added to the diet, not only to supply energy and fuel, their prime functions, but also to spare protein as a source of energy. Very much less protein is needed when adequate amounts of fats and carbohydrates are given. The principal function of protein is that of tissue building.

Various foods furnish different proportions and different kinds of

proteins; that is, proteins supplying different amounts of amino-acids. These will be discussed later. The most important amino-acids concerned in metabolism during infancy are abundant in human milk, less so in cow's milk, and far less in vegetables. The nutritive value of the protein content of diet is judged by its yield of the essential amino-acids. These acids are synthesized into protein which is built up into body tissue.

There has been much discussion as to the protein needs of the adult, particularly as to whether the adult needs more vegetable or animal protein for body maintenance, and this is far from settled. It is also not settled what the total quantity of protein needed by the adult is. The requirements, however, for the growing child have been fairly well established both as to kind and total quantity.

The nursing infant averages from 8 to 12 grams of total protein intake daily. When this is computed according to weight and caloric requirement it will be found that the equivalent is about 1.5 gram per kilogram, and this may be taken as the minimum requirement for the nursing infant. This intake is maintained up to about the sixth month. This is an apparently small total amount, but it shows that the protein of human milk is best adapted in its amino-acid content for digestion and assimilation by the infant. Lactalbumin, which is present in human milk to the extent of two-thirds of the total protein content, promotes growth.

When cow's milk is substituted for human milk in the infant's dietary, the protein intake must be vastly increased, doubled or even trebled. The artificially fed baby on the usual dilutions of cow's milk gets from 15 to 30 grams of protein, which, when appraised according to weight, will be found to be equivalent to from 3 to 4 grams per kilogram of body weight. This will be seen to be a great increase over the amount received when human milk is fed. This increase in the protein requirement over human milk when cow's milk is taken is due to the difference in the amino-acid content of the respective proteins of the two milks. We have seen that the lactalbumin of human milk is the predominant protein, while the predominant protein of cow's milk is casein, and is present to the extent of five-sixths of the intake. This casein does not promote growth to the extent that lactalbumin does.

With certain newer developments in infant feeding, especially the administration of undiluted cow's milk which has been acidified with lactic or other acids, it will be found that many infants take a much



larger amount of protein than what has been mentioned without apparent injury. When observations have been made for a sufficiently long time on this form of feeding and other diets not only high in protein content but of a uniformly high caloric equivalent, our ideas may have to be revised.

The importance of protein in the diet of infants and older growing children may be very forcibly demonstrated when a diet deficient in protein is prolonged. Prolonged deficiency results in marked retardation of growth. This retardation may be only temporary, provided an adequate supply of protein is administered before irrevocable harm is done. If the deficiency is delayed beyond that period, however, the retardation is apt to be permanent. Anemia is an almost invariable result of a prolonged diet poor in protein content. Poor circulation, as shown by cold extremities and mottling of the skin; feeble muscle power and a general disinclination to exertion are a part of the same symptom-complex and are often accompanied by various functional nervous disorders. These symptoms are a part of a state of general malnutrition due not so much to an inadequate supply of food as to an improperly balanced ration. The fat and apparently well-developed child who has been fed on a too large proportion of carbohydrate, furnishes a good example of this condition.

Protein in the form of casein in excess of the body requirements produces large dry stools, which are usually alkaline, due to a large proportion of mineral salts. This state in infants has a distinct therapeutic advantage in certain forms of diarrhea, especially the fermental type.

*Protein Needs in Older Children.*—When we turn from the infant to the older child we find again that protein is by far the most important of the food elements. Estimations of the requirements of the older child are not so easy to make as are those of the infant. As a rule, they are made by taking large numbers of children in groups and averaging their intake over long periods of time. In the second year it will be seen that the average protein intake increases materially over that taken by the nursing infant or the one fed on modifications of cow's milk. During the second year the protein intake averages 44 grams per day, and this well represents the need for that period. This figure rises gradually during the next few years until during the sixth and seventh years, which are years of increased activity, when the requirement will be found to be 60 grams per day. From this time on there is again a steady rise until the requirement in protein reaches 90



grams per day at the twelfth year. Between the twelfth and fourteenth years, the period of sex development, the need is 130 grams per day. From now on there is a sharp decline in protein need and we find that at eighteen years of age the average person requires only 115 grams per day. This closely approximates the adult average, not taking into consideration vocational needs. From one year of age throughout childhood, about 15 per cent of the total calories taken in the daily dietary should be in protein. The above figures when transposed into a quotient will be found to be, on an average, very close to the following: from the end of the first year to the sixth year, 3 to 4 grams of protein per kilo of weight per day, while those over six years will be found to require little if any over 2 grams per kilo per day.

These figures represent the average requirement of the child who is well developed and well nourished and who leads a life of normal activity. Very active children and those who are malnourished or poorly developed need more than the amount stated. The amount required in excess of these figures depends on circumstances, such as the degree of activity, malnutrition or underdevelopment. The exact requirement in a given case may be expressed in terms of what the average normal child requires at a given age or height. The sources of protein in older children are two-thirds animal and one-third vegetable.

**Fat.**—The rôle of fat in food and in human economy has not been determined with the degree of exactness that protein has. On this account it is not possible at present to determine to what extent the body needs fat; that is, in what quantities it should be taken. That it is an important element in food and that the body requires a large amount of fat in the dietary, especially during infancy, is suggested by the large percentage of fat in milk. The early conception with regard to the relation between fats and carbohydrates was that they were interchangeable in the diet. More recent studies, however, have shown that each has its specific function and in the growing child these two should be administered in a more or less definite proportion. The most important function of fat is as a source of energy. Fat not only supplies energy for the daily needs of the body functions, especially activity, but when stored in the body as fat can be drawn on as needed when the normal supply of food is diminished through illness, especially of the febrile type, when there is increased metabolism, or starvation. In the body of a normal infant it has been found that from 12 per cent to 13 per cent of the total body weight is fat. In atrophic infants

there is marked diminution of both proteins and fats, the latter showing only about 0.5 per cent to 2.0 per cent of total weight.

But fat has other and important functions in the dietary than merely as a source of energy. Fats are the principal conveyors of the fat-soluble vitamin A. In human milk the amount of the vitamin is usually adequate; hence, we may disregard this need so long as the child is on breast milk, though the diet of the mother determines to some extent the vitamin content. The average diet of a nursing mother usually provides for this need amply. Infants fed on cow's milk, on the other hand, should be watched, since the vitamin content of cow's milk varies definitely with the food on which the cows are fed. For example, pasture-fed cows produce a milk containing a much larger amount of vitamin than those fed on dry provender. Thus, summer-fed cows produce a milk superior in vitamin content to that of winter-fed cows. Provender which has been dried and, therefore, necessarily aged, is known to be deficient in vitamins. A diet of milk from poorly fed cattle may produce an almost complete avitaminosis.

Fats apparently have a definite and an important function in promoting mineral metabolism, particularly that of calcium. It has been stated that fats tend to prevent a susceptibility to infections, notably tuberculosis. Fats have the function, moreover, of sparing protein to a certain extent, but in this respect are far inferior to carbohydrates.

The caloric value of fat is more than twice as great as carbohydrate or protein and is, therefore, in this respect a most valuable article of diet.

The fat requirement of infants is high. The exact amount varies according to circumstances for the first few months, but by the fourth or fifth month the nursing infant gets from 35 to 40 grams per day, which averages about 4 grams per kilo of body weight. Thus, fat supplies over one-half of the total calories of the daily diet. The daily requirement of 4 grams per kilo of body weight gradually declines until from two to four years the requirement will be found to be about 3 grams per kilo of weight. This amount is sufficient to supply energy, spare protein and insure the absorption of calcium. In older children, in fact throughout the rest of the period of growth and development, the need is met by 3 grams of fat per kilo per day. After the age of infancy, the fat need is met by either animal or vegetable fat.

**Carbohydrate.**—The function of carbohydrate is twofold. First, it supplies energy for muscular activity, though the energy needs of all the body cells may be furnished by this food element. The second

important function of carbohydrate is the supplying of heat to the body. Since energy implies oxidation, oxidation implies heat; therefore, both heat and energy are derived from the oxidation of carbohydrates. When the store of glycogen is drawn upon in the course of muscular activity, it is replenished by the ingested carbohydrate. In some respects fats and carbohydrates are interchangeable, but only to a limited extent. Both supply energy, fat to a lesser extent than carbohydrate. Some authorities consider carbohydrate as the only source of energy.

Carbohydrate spares or protects the body protein through its own oxidation. It accomplishes this by supplying a substance which is more easily broken down for energy-yielding purposes. This function is shared by fat, though in this respect carbohydrate is superior to fat. It cannot, however, entirely supplant protein. The carbohydrates do not add to the structural maintenance of the body. When carbohydrate food is increased in the daily dietary to a maximum of safety, the protein can be reduced to a point where only structural maintenance is taken care of. Carbohydrate is necessary in the food in certain quantities, for if it is not furnished the body breaks down its protein in order to supply its carbohydrate needs. Fats cannot replace carbohydrates in the dietary, but carbohydrates can replace fat and, when taken in excess beyond the power to convert them into glycogen, form fat. This is frequently observed in the so-called sugar baby and some of the obese children of a later period. Carbohydrates may be considered as participating in structural processes, but only to a slight degree. Whether carbohydrate utilized thus is derived from the ingested food directly or from other sources is not as yet determined.

In addition to those functions already enumerated, carbohydrate is necessary for many processes of metabolism. Fat cannot be completely oxidized unless a certain amount of carbohydrate is undergoing simultaneous oxidation. When carbohydrate is absent from or deficient in the diet imperfect oxidation of fat occurs, leaving for elimination incomplete products of fat metabolism—diacetic and  $\beta$  oxybutyric acids, known as the keto-acids. Thus there may be produced a depletion of the alkali reserve of the body fluids, with a resulting acidosis.

Carbohydrates promote normal body growth, but only by supplying a large part of the energy requirement. They perform an important function by adding to the body weight, which they do by promoting the storage of glycogen and the deposition of fat. They aid further in the increase of weight by promoting water retention, without which

gain in weight is impossible, and in fact in many instances gain in weight cannot be effected without the aid of carbohydrates.

*Requirement.*—Nursing infants average 12 grams of carbohydrate per kilo per day. Artificially fed infants average more than this and often get far more than their body requirements. In infancy carbohydrate is taken in the form of soluble carbohydrate, that is, sugar. The sugars generally used for this purpose are: lactose, exclusively in the breast-fed infant, while the artificially fed infant receives its sugars as lactose, cane sugar or maltose, or generally the dextrin and maltose mixtures. Children who have passed the nursing stage and are on a general diet average 10 grams of carbohydrate per kilo per day. Unlike the nursing infant, they receive their needed carbohydrate in the form of soluble carbohydrate (sugars) and insoluble carbohydrate (starch), in about equal proportions, or, to be exact, 49 per cent sugar and 51 per cent starch.

Because of greater economy both commercially and physiologically there is an increasing tendency to the use of carbohydrates in the dietary of older children to the exclusion of protein. There is, of course, as may be readily observed a distinct danger in the neglect of protein in the diet of a growing child. If too large amounts of carbohydrate are taken, the intake of both fat and protein necessarily suffers. Because of this, an excessive intake of carbohydrate causes an abnormal deposit of fat, without promoting muscle development. The result of this is that we have an abnormally fat child with a feeble resistance.

Carbohydrate is useful in supplying the needed calories when the quantity of protein and fat present in the food is sufficient and more calories are needed. Thus, increased caloric need *due to activity* may be supplied by carbohydrates alone. This is a very important fact, especially when dealing with the undernourished child. The total requirement of the three food elements which have been discussed as measured in caloric value is in the following proportion:

Proteins .....	15 per cent of the total calories
Fats .....	35 per cent of the total calories
Carbohydrates .....	50 per cent of the total calories

**Mineral Salts.**—In the early studies of metabolism little or no attention was paid to the mineral salts, consequently they were imperfectly understood. They are even yet not understood as well as are the other food elements which have just been considered. There are certain conditions under which these salts must be studied which



render their exact determination difficult. Some of them are found in the food in inorganic form and some in organic form. Regardless of the form in which they are ingested they are for the most part excreted as free salts. Salts may function as free salts or in combination with organic compounds in the various processes of metabolism. Regardless, however, of the form in which they have thus functioned, they are broken down finally into their inorganic form. Still other mineral substances are supplied in the food either in organic or inorganic form, are metabolized, and are excreted in either form.

The mineral matter of the food which the human organism ingests, the total amount of mineral contained in the body, and the amount of mineral material excreted can be measured only in the form of ash. We can see at once what difficulties present to an accurate determination of these substances quantitatively, when we consider such a method in comparison with the facts mentioned in the preceding paragraph. In spite of all difficulties, much has been learned regarding the total quantity of the mineral constituents of the several body tissues, and also the function of many of the inorganic elements in vital processes.

When we considered protein and its functions, the statement was made that with the exception of the mineral salts which take part in the development of skeletal tissue it was the most important of the food elements. It is obvious that such an exception must be made since the human body could not function without its skeleton.

The mineral elements are therefore essential to life, and in a very important way, and yet are not essential in the same manner that the so-called food elements (protein, fat, and carbohydrate) are. Normal bone consists of one-third organic and two-thirds inorganic material. Of this inorganic portion, the main constituent is calcium phosphate with a small portion of calcium carbonate, the former existing to the extent of 85 to 90 per cent. The organic compounds composing the solid portion of the soft tissues depend for their composition and function upon the mineral salts. Their importance in both respects is demonstrated by animal experimentation, when it is shown that when fat, protein, carbohydrate and vitamins are supplied in adequate amounts, both growth and weight cease if calcium and sodium chlorid are lacking. Mineral salts play important parts in such functions as secretion, excretion, absorption, and retention. All of these functions are essential parts of the metabolic processes and in this way the salts become necessary. They play an important part in the acid-



base equilibrium of all body tissues. The very important function of osmosis is determined by the solvent power and reaction of certain fluid tissues and is controlled largely by mineral salts and protein. Both calcium and sodium, but particularly the latter, promote water retention.

Certain salts, notably calcium, and to a lesser extent sodium, influence the elasticity of muscle and the irritability of nerve. Fat, as we have seen, promotes the absorption of calcium and phosphorus and probably their retention also. This has an important bearing on certain metabolic processes, notably that concerned with the development of rickets. The salt balance of the body is easily disturbed, especially in infancy and childhood, most frequently during acute illness, particularly of the diarrheal type, and after anesthetics. When this balance is disturbed to any large extent serious conditions are produced.

**Water.**—No accurate determination has yet been made of the exact requirement in fluid intake for the human being. A large amount of water is contained in the usual daily diet and is probably regulated by natural processes to a fair extent, in order to meet the minimum requirements of the adult, with a relatively small amount of added water. In adults the amount of water to be added depends on many factors. The character of the food, whether relatively high in protein, carbohydrate or fat, will determine to some extent the need for additional fluid. The preponderance of leafy vegetables or food of a drier nature will have a distinct influence. Exercise, which increases elimination through skin and lungs in an effort to maintain body temperature, naturally calls for increased water intake. On the other hand, persons leading a sedentary life, and eating a meager amount, need relatively little additional fluid. The temperature of the surrounding atmosphere, particularly incident to the various seasons, largely determines the relative amount of elimination of water through the skin or kidneys. It will be readily seen, therefore, that it is difficult to calculate with any fair degree of accuracy what the requirement in fluid intake should be.

In infancy, on the other hand, some estimate of fluid needs has been made. The food of infants is composed largely of water; in fact the water content varies from 85 to 90 per cent of the total amount. The sugar, salts, and some of the proteins are ingested in solution, while other proteins, and all of the fats are in the form of suspensions or emulsions. In addition to this, water is needed as a food diluent for proper metabolism of all foods. More particularly at this time than later, water is needed for elimination, absorption, and retention. The variation of water intake largely determines the fluctuations of daily

weight in young infants, gain in weight at this period being largely a matter of water retention. The amount of water retention in turn is determined by the salts, particularly sodium chlorid. The retention of water is also greater when carbohydrate predominates in the diet. A large amount of urine of low specific gravity is characteristic of infancy and early childhood. For this reason and from the discussion immediately preceding it is obvious that the water intake should be in fairly definite quantities, the amount being of vast importance.

Investigations have shown that in early infancy the need of water is approximately one-fifth of the body weight. From the third to the sixth month the requirement is about one-sixth of the body weight, while by the end of the first year the requirement is reduced to approximately one-eighth of the body weight. Expressed differently, for the first six months of life, the requirement is somewhere near two and a half to three ounces per pound of weight per day; while for the second half of the first year, from two to two and a half ounces per pound per day will usually suffice. After this time, the requirement and intake vary according to the same circumstances as in older children and adults. These figures represent the requirement in normal well-developed and nourished individuals. Dehydration due to disease, digestive disturbances, and starvation, requires amounts dependent on the condition, and often far in excess of the figures stated. The water ingested is disposed of as follows: 59 per cent is eliminated by the kidneys; 33 per cent by the lungs; and 6 per cent by the intestines; while only from 1 to 2 per cent is retained. This last fact, as we have seen, is dependent on many conditions, such as the amount of carbohydrate in the food, the amount of salts, particularly of sodium chlorid, and other dietary factors. The amount of retention stated, of course, applies only to health; it is modified in both directions by many diseases and states of nutrition.

**Vitamins.**—In the course of food experiments with animals it was found that when these animals were fed an artificial diet adequate so far as protein, fat, carbohydrate, salts, and water were concerned, they either failed to become properly nourished, or in the case of young animals, failed to grow or develop in a normal manner or even perished. In some instances they developed symptoms which were closely analogous to certain well-known symptom-complexes in human beings. It was recognized at once that there was something still lacking in the food which was essential to proper metabolism and development. Two of these conditions had been studied in the human being, but in an

entirely empirical manner. It had been observed for some time that scurvy developed on a diet which lacked fresh vegetable or fruit juice and that beriberi developed on a diet consisting largely of rice which had been prepared in a certain manner. It had already been observed that the juice of citrous fruit or the juice of certain vegetables prevented or cured scurvy. Likewise preparations of the polishings of rice accomplished the same in beriberi. These two conditions were produced artificially in animals and birds, through deliberate administration of foods which were known to be lacking in certain food elements.

The result of such observation was the discovery of certain elements of food to which a number of different names were given according to the observer or his point of view. Among the terms applied to these substances were "nutrient principles," "stimulants," "determinants," "food factors," "accessory food factors," etc. On account of the importance attributed to the amino-acids in growth and development, the name vitamin was finally applied to these newly recognized substances implying "life-giving amines." The term preferred by many investigators is "food hormones."

Vitamins have been variously classified according to the substances in which they are soluble, such as "fat-soluble vitamin" or "water-soluble vitamin," etc., or according to the disease or condition with which they were first found to be associated, such as "antiscorbutic vitamin," etc. The terminology at present most in use is to designate them by letters, such as vitamin A, vitamin B, etc.

Vitamins have not been isolated in pure state, nor have they been accurately classified chemically or biologically, but they can be separated from the food in which they occur, and chemical formulas have been furnished by several laboratory workers. The accuracy of these is yet to be proved. A great deal is known concerning three vitamins and from what foods they are derived, and in what quantities they are present in these foods. It is likely that the existence of two other vitamins has been demonstrated. There can be little doubt that many more, concerning which we as yet know nothing, will eventually be discovered. That vitamins are present in sufficient amounts in the food generally consumed is to be supposed, else so-called deficiency diseases would be far more common than they are. They are present even in a balanced ration in relatively minute quantities and, therefore, do not supply energy, nor directly participate in tissue building, but they are necessary to promote tissue growth and maintenance. They are,

therefore, "factors which probably activate certain tissues to perform a definite function in metabolism."

*Vitamin A.*—This vitamin has also been called the "fat-soluble vitamin" because it is very much more soluble in fats of various kinds than in any other medium; in fact, it is conveyed only in the fat of foods. It is also called the anti-ophthalmic vitamin. Its presence in large amount in cod-liver oil has brought this substance into prominence as a therapeutic agent and has made it almost synonymous with the vitamin. This vitamin also occurs in the fat of milk (cream) and in butter itself. Certain parenchymatous organs of many animals contain it to a considerable extent as does the yolk of egg, and a small portion is found in certain other fats of animal origin. It is not found, however, generally in animal fat. Vegetable fat does not contain the substance, but certain leafy vegetables available for human economy contain sufficient amounts to make them valuable as foods for the vitamin content if not for other reasons. Notably among these are cabbage and spinach. Alfalfa is conspicuous among foods for cattle and other animals for its vitamin A content.

This vitamin is known for its growth-promoting properties, which function it shares with other vitamins and is important especially during the period of growth and greatest development. It has long been known that cod-liver oil is useful in the treatment of rickets, and there has been repeated effort to connect this function of the oil with vitamin A. Such a connection has apparently recently been established in the discovery of vitamin D. Certain ophthalmias (xerophthalmia-keratomalacia) have been shown to be definitely associated with deficiency of vitamin A, and are prevented, or cured, as the case may be, by its administration. It is probable that other food conditions play an important part in these eye disturbances and that the vitamin alone cannot be held responsible. A number of other tissue changes, possibly degenerations (hornification), have recently been shown to be connected with vitamin A deficiency.

*Vitamin B.*—Vitamin B is so intimately associated with studies in beriberi that it is also called antineuritic vitamin. Historically, our first knowledge of this substance dates from observations made on food as an element in the cause and prevention of beriberi in the enlisted men of the Japanese navy. To be sure, no knowledge was possessed at that time as to the nature of the substance as we know it to-day as a food factor, but the character of the food was studied, or at least observed to an extent which showed that certain foods or groups of



food appeared to be directly connected with the development of the ailment, while other foods either cured or prevented the polyneuritis. These observations were followed by others in different localities with much the same conclusions, and more or less definitely accepted as a result of observations on the diet of the "Philippine Scouts" under the United States Army Medical Commission.

It was not until the identity of polyneuritis or beriberi in man, and the polyneuritis of birds was established that the theory of food factors was definitely accepted as the etiological agent. The studies on this vitamin have been so closely associated with the polishings of rice that it was thought for some time that rice diet, either polished or unpolished, was the sole consideration in its cause or cure. Subsequent studies, however, have shown that in all probability there are a number of other food elements to be considered. The most convenient and easily available food for study remains the polishings of rice. A prolonged diet of white bread which contains little or no yeast will also produce the disease and this fact led to the discovery that yeast is a very fertile source of the factor. A large number of ordinary foods, including milk, fruits, all of the leafy vegetables, some of the other vegetables, and to a less extent a number of animal foods, contain sufficient amounts of this vitamin to render a deficiency of it on simple balanced diets entirely unlikely. It has also been shown that this vitamin strongly stimulates the appetite. This vitamin shares with vitamin A the function of growth promotion so that to a large extent they are interchangeable for this purpose.

*Vitamin C.*—Vitamin C like vitamin B is definitely associated with a single disease—scurvy. It was known for a long time that scurvy could be prevented, and cured after development, by the administration of the juice of certain vegetables and fruits. At a much later date it was found that the antiscorbutic properties of certain foods were due to a definite substance known as vitamin C, or antiscorbutic vitamin. This furnishes one of those striking instances in medical knowledge of empiricism preceding scientific discovery. Vitamin C is most abundant in the citrous fruits and next in the tomato. It is found also in lettuce and to a less extent in certain other vegetables. Milk contains this factor in varying amounts, but usually to an extent, in fresh milk, sufficient to protect a milk-fed infant from active scurvy. The amount of this vitamin contained in milk depends on the provender on which the cows were fed. This statement applies equally to the vitamin content of all milk. Prolonged heating, evaporation or pasteurization, and



aging, either with or without heating, impairs the vitamin of milk. Rapid heating apparently destroys very little of the contained vitamin. Preparation of certain vegetable foods by prolonged boiling, and some canning processes, also lowers the vitamin content. In some instances aging and not heating is the vitiating agency. The studies on vitamin C have probably done more than any other studies to identify infantile scurvy with the adult type of this disease.

*Vitamin D.*—For a long time it was suspected that vitamin A was the antirachitic factor contained in cod-liver oil. This idea received support from clinical evidence. More recent studies have shown that cod-liver oil produces certain results when given whole (the cure of xerophthalmia) and aided in the cure of rickets. It was further found that the cure of rickets was effected or rather promoted by the administration of cod-liver oil from which the vitamin A had been removed. This led to still further experiments which have tended to show that there really does exist in cod-liver oil a substance which promotes the deposition of calcium and possibly also the retention of phosphorus. This is entirely distinct from vitamin A, and has been given the name of calcium depositing substance, or vitamin D.

*Vitamin E.*—Evidence is at hand which tends to prove that a vitamin exists which determines fertility, at least in rats. One of the theories adduced in explanation of the influence of this vitamin is that when the female rat is deprived of it, an anemia of certain portions of the ovum develops. It is not likely that this is the only explanation, since infertility develops when only the male rat is deprived of the vitamin. This substance is found abundantly in the wheat germ and has been given the name vitamin E or vitamin X.

Since we know definitely the function of several substances or factors, to which the term vitamin has been applied, and since by analogy there are a number of metabolic changes in the body which suggest dependence on vitamin stimulation, it is likely that more vitamins exist than have as yet been found and that the possibility of the number of these substances is large.

That these substances are of vast importance cannot be denied, and yet their distribution in natural food is varied and extensive. In a well-balanced diet, therefore, it is not necessary to add artificially prepared vitamins to those already present. These substances are of great importance, as all of the evidence at hand attests. Their distribution in nature is extensive and in most of the foods they exist in sufficient quantities.

**Sunshine.**—The fact that sunshine is necessary to all forms of plant life with the exception of a few species of very low order, has been recognized for a very long time. In most forms of plant life the metabolic processes cannot take place unless the plant is exposed for the greater portion of the time to the rays of the sun. Particularly is this true so far as the absorption of water is concerned, and also the discharge of that portion of the absorbed water not needed for metabolic purposes, or what has been utilized and is not further needed. Transpiration cannot take place without water. Practically all of the phenomena of plant life and development are dependent on this food element, a fact so well recognized as to be common knowledge.

The essential part played by sunlight in the phenomena of life, growth, development, and metabolism as occurring in animals appears not to have been appreciated even partly until comparatively recent times. That sunshine has a definite influence on certain body tissues, in both health and disease, and that all of the metabolic processes, especially those of the mineral salts, are influenced by sunlight has been demonstrated with striking vividness during the recent past.

What particular ray or combination of rays found in the solar spectrum furnish the potency of sunlight in influencing any or all of the phenomena of life is not clear at present; but enough is known about the action of the ultraviolet rays to make it reasonably certain that a large influence is exerted by them. They too have been studied carefully under conditions rendering their control accurate, and so for all practical purposes when discussing sunlight in regard to physiological activities, and also in therapeutic application, the ultraviolet ray or ultraviolet radiation is referred to.

Ultraviolet radiation probably has a definite influence on all metabolic processes; but the influence of these rays on mineral metabolism has been studied more extensively than that of other food elements and most of this work has been done on a single disease—rickets. Ultraviolet rays promote the absorption and retention of both calcium and phosphorus. On exposing the body to ultraviolet radiation the skin becomes fluorescent (hematoporphyrin). Then follows a dilatation of the superficial capillaries and those of the subcutaneous tissues, possibly due to an irritation of the sensory nerves by the fluorescence or the direct action of the rays themselves. It has been suggested by way of explanation that certain changes occur during this process which produce substances which, when absorbed by the general circulation, affect the metabolism directly, or act in a therapeutic manner through bacte-

ricidal action in the intestines and thus prevent the bacteria from exerting a deterring effect on the absorption of food. The specific action of the rays on the absorption of calcium is explained by their effect on cholesterol, a substance occurring plentifully in the skin, whereby a change is effected which enables it to develop antirachitic powers. How this power is formed or transmitted is not known; it may be that the altered cholesterol becomes endowed with vitaminic power, or it may become radioactive and be absorbed and circulate as a radioactive substance. This discussion is alluded to further in the chapter on Rickets.

It is claimed that in the process of ultraviolet radiation of certain of the lower animals a stimulation of the endocrine glands occurs, causing a hyperplasia. This hyperplasia is particularly noted in the parathyroids, which are known to influence calcium metabolism.

That alterations of various body tissues take place in the course of ultraviolet radiation is well known. The coagulability of the blood is increased, the red blood-cells are increased in number, as are the platelets, while there is a constant and uniform leukocytosis following radiation. The bactericidal action of the rays on infectious organisms within the body is claimed by some, the bacteria resisting power, or even an actual bactericidal action of the blood, being the explanation. None of these statements has been actually proved; the infection resisting power given to the tissues by the rays may be due to the other changes in the tissues, especially the blood, as just described, rather than to a specific power.

Whatever the specific influence of sunlight on the metabolic processes, its influence is great and essential. A striking analogy appears between sunlight and the vitamins, and this agent must be placed among the food factors.

## SOURCES OF FOOD ELEMENTS

The part played by the various food elements in the human economy has been covered in the preceding pages. It is the purpose of this chapter to discuss briefly the sources of the various food elements as found in the normal dietary of infants and children. For convenience, foods will be divided into protein foods, fat foods, carbohydrate foods, and foods furnishing an abundance of salts and vitamins. Milk cannot properly be classed under any of the above headings and will be discussed under a separate paragraph.

**Milk.**—Both human and cow's milk may be considered as the only complete food, in that milk furnishes sufficient protein for growth and development, fat and carbohydrates in generous proportions, and liberal amounts of the necessary salts and vitamins. There is a marked difference, however, both quantitatively and qualitatively, between the various food elements as found in cow's milk and in human milk. It is in the proteins that this difference is the most marked. Cow's milk contains more than twice as much protein as does human milk. This explains the dilution of cow's milk in infant feeding and the addition of carbohydrates to make up the low caloric value resulting from such dilutions. Not alone does the quantity of protein of the two milks differ, but there is also a difference in the quality of their respective proteins. In human milk, the soluble or whey proteins are equal to or in excess of casein or coagulable protein. In cow's milk the casein is three to five times that of the whey protein.

Both milks contain fat in about the same proportions. Here again they differ as to the quality of fat, cow's milk containing about seven times more fatty acids than does human milk.

Carbohydrate is found in both milks in the form of lactose or milk sugar, human milk containing one and one half times as much of this particular substance as does cow's milk. The two milks show great deficiency in their salt content. The salt content of cow's milk is greater than that of human milk. It contains a relatively larger amount of calcium and phosphorus and a smaller amount of potassium and iron salts. Both milks have a low iron salt content, and the results of this are seen in cases of so-called milk anemia occurring in infants fed on milk alone for too long a period.

Fat-soluble vitamin, or vitamin A, is abundantly present in both human and cow's milk. Water-soluble vitamin, or vitamin B, and the antiscorbutic vitamin, or vitamin D, are present to a lesser extent.

**Protein Foods.**—By far the greatest part of the protein of our diet is obtained from the animal kingdom. Foods which are properly classed as protein foods are: milk, eggs, meat, fowl, fish, and certain of the vegetable foods, particularly the legumes. The grains, such as wheat, barley, oats, corn, etc., contain a fair amount of protein. Carbohydrate predominates in such food, however, and for this reason they are properly classed among the carbohydrate foods.

Certain products prepared from meat, such as beef juice, beef extracts, gelatin, etc., are more easily digested than is meat itself and for this reason are useful in various illnesses when an easily digested



protein food might be desirable. The nutritive value of such preparations is far less than that of meat itself.

Egg white is the part of the egg which is rich in protein, while the yolk is rich in lipins or fats. Eggs, as such, are not only important in a child's dietary, but are essential in the cooking of many preferred foods, such as custards, spoon bread, etc. There are on the market certain proprietary foods high in protein content, such as powdered protein milk, and the various preparations made rich in protein by the addition of dried calcium caseinate obtained from milk.

**Carbohydrate.**—For the first few months of life, carbohydrate is supplied by lactose of human milk and cow's milk, and those infants artificially fed receive carbohydrate in the form of added sugar. The sugars commonly used in infant feeding are sucrose, lactose, and the various preparations containing dextrin and maltose almost exclusively. In the latter part of the first year and from that time on, much of the carbohydrate is supplied in the form of starch. Common articles of diet supplying carbohydrates, other than milk and the simple sugars, are the grains and products prepared from grains, fruits, and potato. The larger part of the carbohydrates in a child's diet is furnished by such foods as cereals, breads, macaroni, rice and fruits. The so-called breakfast foods are for the most part proprietary foods and are nothing more than specially prepared grains. The cereals which necessitate preparation in the home, such as cream of wheat, farina, oat meal, etc., are to be preferred to the ready cooked preparations to be found on the market.

**Fat.**—Most of the fat ingested by the body comes from the animal kingdom, although a very appreciable amount is of vegetable origin. Foods furnishing a liberal amount of fat are milk, eggs, meat, fowl, cottonseed, olive, cod-liver oil.

**Salts, Vitamins, Water and Bulk.**—There is a group of food products valuable because they are rich in inorganic salts and vitamins. Of this group the most important are the green vegetables and fruits. Such foods serve an additional purpose in that they leave considerable residue after digestion, thus providing intestinal peristalsis. Green vegetables and fruits are especially rich in iron; in fact they form the chief supply of iron to the body, they are therefore particularly valuable to the growing child.

**Vitamin A.**—Cod-liver oil is the richest known source of vitamin A. It is said to contain two hundred and fifty times as much of this vitamin as does butter fat. Other fats rich in this vitamin are milk fat



and egg-yolk fat. Fresh green vegetables, particularly the leafy parts, are excellent sources of this vitamin. Spinach, lettuce, and cabbage contain considerable amounts. To a lesser extent it is found in carrots and sweet potato. Tomatoes contain it in abundance. Green peas are said to be rich in this particular vitamin. Fruits contain it only in small quantities. The milk from grass-fed cows is rich in vitamin A, while stall-fed cows produce a milk low in this particular vitamin.

*Vitamin B.*—Vitamin B is widely distributed in our foodstuffs. It is abundant in green vegetables. Cereals contain it in an appreciable amount and tomatoes are particularly rich in it. Fruits contain vitamin B in abundance.

*Vitamin C.*—The following foods are valuable sources of this particular vitamin: lemons, oranges, bananas, tomatoes, cabbage, lettuce, spinach, beans, peas, and turnips. Grains, as a class, contain little or none of it. Meat contains very little. Milk contains it to some extent, but is an uncertain source. Raw milk contains a sufficient amount to protect infants against scurvy. This vitamin is easily destroyed by heat and for this reason is either absent or present in only a small amount in certain of the so-called proprietary foods.

*Vitamin D (Antirachitic).*—Like vitamin A this vitamin is fat-soluble and is found in its greatest concentration in cod-liver oil. It has also been found in egg-yolk and milk. Spinach and lettuce have no antirachitic properties. We know but little as to the general distribution of this particular vitamin.

### CHAPTER III

#### DIGESTION AND METABOLISM

We have considered the various food elements and their relation to the human economy. In order to fully appreciate the part they play in tissue building and in nutrition and growth generally one must know the processes of digestion and subsequent metabolism. In infancy, certainly during the early months of life, the food consists naturally of milk from the mother. Fortunately, this food has the various elements so accurately adjusted to the needs of the organism that no particular care need be taken regarding anything but the total quantity. Even when a substitute milk is administered, modern methods of adaptation have eliminated much of the necessity of readjustment beyond dilution, and the addition of lacking carbohydrate. Neutralization of the buffer substances in cow's milk through acidification has still further simplified adaptation. Later in childhood and throughout adult life more attention must be paid to the balance of food elements, bringing the ratio of the several elements in accord with the needs for these substances in the various processes of development, growth, and activity. A brief summary, therefore, of digestion and metabolism of food elements as a step in appreciating their relation to development follows.

**Fat.**—In infancy all fat is ingested as neutral fats. In older children and adults fat is ingested in other forms than neutral fats as well. On entering the stomach fat is enmeshed in the curd and this is practically the only preparation it undergoes at this point. There is a slight action of lipase in the stomach, but it is doubtful if there is much change by this enzyme, since the acid reaction of the chyme checks its activity. Fats enmeshed in the curd, which forms in the stomach, pass on into the intestine and are slowly liberated from the curd and are acted on by the digestive ferments of the intestinal tract. The first change which takes place in fats is that of emulsification, which process furnishes increased surface for enzyme action. They are then split into fatty acids and glycerol by lipase from the pancreas aided by the bile salts, glycocholate and taurocholate of soda. A part of the fatty acids formed is saponified by the alkaline carbonates of the intestines and aids in the

further emulsification of the fats. Fatty acids and soaps are absorbed as such by the epithelium of the villi where they are resynthesized back to neutral fats. This is transferred to the lacteals but the processes involved are not definitely known. Not all of the fat ingested is absorbed even though it has gone through the changes described. The unabsorbed fat and fatty acids are excreted. Unsplit fat is not absorbed by the lacteals, as such, but is changed back in its passage through the intestinal wall to neutral fat by lipase, which has the power of reversing its action. All fat found in the lymph stream is in the form of neutral fat.

During health, in both breast-fed and artificially fed infants, the proportion of absorption of ingested fat is high: about 90 per cent in the former and somewhat less in the latter. During the latter part of infancy, the proportion of absorption decreases slightly and continues to decrease, though not to the same extent, during the rest of childhood. In the course of all illness, but especially that due to infections, there is a marked diminution of all digestive ferments but particularly the fat-digesting ferments. In the course of diarrhea there is not only a diminished secretion of enzymes, but also a rapid elimination. During illness, therefore, the absorption of fat is diminished, with the result that there is more fat found in the stools. The amount of fat absorbed and the amount found in the stools, either as soaps or undigested fat, depend largely on the amount ingested. If the amount ingested is just sufficient to meet the body needs, the larger proportion is absorbed, and only a relatively small amount is found in the stools and this in the form of soaps. Almost no unchanged fat is found in the stools under these conditions. When the amount of ingested fat is in excess of the body needs, a relatively large amount of soap will be found in the stools, and the resulting dejecta are constipated.

In those instances where fat has been ingested in quantities far in excess of the body needs, especially where this has continued for prolonged periods, free fat globules are found in the stools and these stools are loose, and are in reality a true fat diarrhea. Of course, in order for this to occur, the fat ingested must not only be more than the body needs but must be also in excess of the power of the digestion to care for the extra load.

Chronic malnutrition, particularly in the infant, either accompanies or produces a fat intolerance. As a result, the ingestion of fat, even to a small extent beyond the tolerance, results in an excessive excretion of many of the mineral salts. Some of this intolerance is probably

due to the fact that in malnutrition there is a diminished secretion of all enzymes, but particularly the fat-splitting ferments. The so-called exudative diathesis is also accompanied by a fat intolerance, whereas the carbohydrate tolerance is either normal or in some instances is apparently increased.

The transportation and deposition of fat after leaving the thoracic duct is an important phase of our studies and for a long time accurate observations were few, and misleading statements many. Considerable progress, however, has been made in our knowledge on this subject in the last few years. Although fat is found in the thoracic duct only as neutral fat, it is normally transported in the blood, not as neutral fat, but as a complex organic substance, lecithin, whose molecule is a compound of fat glycerol, phosphoric acid, and cholin, which is easily miscible with water. The processes through which this change is effected are not as yet understood, but we do know that this substance is carried for the most part in the red blood-cell. The lecithin is conveyed to the various body tissues where it is either deposited as fat or utilized by the tissues in metabolic processes. Apparently lipases exist in very small quantities in any of the tissues of the body except in intestinal juices, but there are certain tissue enzymes which are capable of breaking up lecithin for metabolic processes.

As we have seen before, the rôle of fat in the body is important and it fulfills this rôle through a series of changes, after absorption, which are in some instances relatively simple and in others quite complex. The main function of fat is as a heat producer. The fat serves in this capacity largely if not entirely through the simple process of oxidation. The storage of fat in the body is second in importance only to its function as a heat producer. Fat is stored in the subcutaneous tissues as a protection against thermic changes and elsewhere as a mechanical protection to various organs and tissues. The fat thus stored is drawn on during starvation and in the course of diseases which are accompanied by a diminished intake of food. Lipase probably controls the output of fat to the blood. The ultimate fate of fat is oxidation into water and carbon dioxid. One of the most important metabolic phenomena relating to fat is the fact that during the process of oxidation in certain conditions, such as starvation, various fevers and diabetes, there are formed the acetone bodies which produce a definite train of symptoms accompanied by acetonuria. Both acetone and diacetic acid are normally foreign to the body. Acetone is eliminated through the lungs and kidneys. Diacetic acid must be neutralized in



order to prevent any marked change in reaction of the blood. This is accomplished by a reserve of alkali present in the blood for this purpose. The neutralized acid is then eliminated by the kidneys. During starvation, in the course of certain fevers, and in diabetes mellitus, a large amount of protein can be changed into fat.

In carnivora, the source of fat in the food is generally derived from fats and carbohydrates, while in herbivora it is practically entirely derived from carbohydrate. The retention of fat in the body to an extent which we term obesity is concerned with an evidently complex process. There can be no doubt that a definite relation exists between the amount of food ingested and the amount of energy expended, mostly through muscular exertion. In fact, the basal metabolism rate is practically identical in those leading sluggish lives and those who do hard work. Hence, we might be led to the supposition that there are familial and individual characteristics which in a measure govern the degree of obesity. Again there is evidence at hand showing that certain disturbances at present attributed to the endocrine system are responsible for an excessive deposition of fat in the body. But our present knowledge does not warrant a positive statement on this subject. Practically in the great majority of individuals, the amount of subcutaneous fat can be controlled almost at will through a measured and balanced diet and a definite regulation of muscular activity.

**Carbohydrates.**—The processes of digestion of carbohydrate are fairly well understood and more is known concerning its metabolism than that of fat; still our knowledge is far from complete. Although a larger number of enzymes are concerned in the digestion of carbohydrate than of fat, the mechanism of absorption is much simpler. The definitely known enzymes participating in the digestion of carbohydrate are five in number. Ptyalin or amylase is the amylolytic ferment found in the saliva and is derived from the secretions of the parotid, sub-maxillary, and sublingual glands. Its function is to convert starch into maltose. There is also a small amount of maltase found in the saliva. A second amylolytic ferment is encountered when the carbohydrate reaches the intestines; none is found in the stomach. This second starch-splitting ferment, known as amylopsin or pancreatic amylase, is derived from the secretions of the pancreas. It has a similar if not identical action to that found in the saliva. The remaining three ferments having to do largely if not entirely with carbohydrate digestion are found in the secretions of the intestines, or the succus entericus. They are: (1) invertase, also called sucrase, which has for its main



function the conversion of sucrose or cane sugar into glucose and fructose, or levulose; (2) maltase, which converts maltose into glucose and glucose; and (3) lactase, which acts only on lactose or milk sugar, converting it into glucose and galactose. Besides these secretions or juices there are two enzymes which function entirely within two important organs of the body, *viz.*, the liver and the muscles. These are liver glycogenase, and muscle glycogenase, and they have apparently the identical action of converting glycogen into dextrose.

The various enzymes concerned with carbohydrate digestion apparently have a specific action on particular forms of carbohydrate, but all of them function with the same end in view: the breaking down of the complex or poly- and di-saccharids into the simple forms or monosaccharids, in which form alone they can be absorbed and prepared for use by the various body tissues. Carbohydrates are ingested as sugars and starches, called soluble and insoluble carbohydrates. Through the process of cooking, starch cells are broken up and become to a degree soluble, and are classed as soluble carbohydrates, yet they are not soluble to the same extent as are the sugars. Uncooked starch remains in the class of insoluble.

Immediately on being taken into the mouth carbohydrate is mixed with ptyalin, which prepares it for further digestion but it remains in the mouth for too short a time to permit any important change to take place. The ptyalin splits the carbohydrate into dextrin, a non-crystallized polysaccharid, and into the disaccharid maltose. Some dextrose is formed because of the presence of a small amount of maltase which splits maltose into dextrose and dextrose. These changes are probably not direct ones, however, for it is likely that several intermediate changes occur during which hydrolytic action plays an important part. No digestion of lactose takes place in the mouth, since lactase is not encountered until the small intestine is reached. On this account no digestion of food in a breast-fed infant takes place at all in the mouth. There being no diastase in the secretions of the stomach, no gastric digestion of carbohydrate occurs, but the diastasic digestion started in the mouth continues for probably an hour or two in the stomach—until destroyed by gastric juice. Before this they are hydrolized with erythrodextrin. They pass out of the stomach for the process of digestion in a more ideal medium.

On reaching the intestines that portion of the carbohydrate (starch) which escaped the action of ptyalin is now acted on by the amylolytic ferment of the pancreas (pancreatic amylase) and changed into maltose

and dextrin. Cane sugar (sucrose) is converted into glucose and fructose by the action of invertase (sucrase) from the intestinal secretion. Maltose is split into dextrose by the maltase, also found in the secretion of the intestine. Now for the first time, that is, in the intestinal tract, lactose is acted on at all. This sugar is changed by lactase into dextrose and galactose. The changes through which the various carbohydrates pass when acted on by the digestive ferments may be expressed in another and perhaps clearer way as follows: Lactose is changed into dextrose and galactose. Saccharose (cane sugar) is changed into levulose and dextrose.

Starch is first dextrinized, becoming amylo-dextrin; this in turn is changed into erythro- and achro-dextrin while this is further changed into maltose, which is finally broken down into dextrose and dextrose. Maltose ingested as such becomes at once dextrose and dextrose which is the most easily absorbed of the sugars. Starch which escapes entirely any action of digestion is passed by the feces as are di- and polysaccharids, neither of which is oxidized or otherwise utilized by the body metabolism. Bacterial action on the carbohydrates through fermentation and other processes plays an important part in the intestinal digestion of this group of food elements.

When the carbohydrates have been hydrolized by the various enzymes into monosaccharids, they are absorbed directly by the capillaries of the villi of the walls of the intestines. The absorbed monosaccharids go directly to the liver by way of the portal vein and are there synthesized in the liver cells through enzymic action into glycogen, a polysaccharid, also known as animal starch. An appreciable amount goes directly to the muscles and is there acted on by muscle glycogenase to form glycogen, and is likewise stored for future use. The carbohydrate content of the general circulation is invariably in the form of glucose and in health this is maintained at a fairly constant level. This function is performed by the hydrolysis of stored glycogen. The liver not only controls the level of glucose content of the blood stream, but is the main storehouse for reserve glycogen. From time to time the stored glycogen is drawn upon and utilized for purposes of energy production in the tissues, passing through several stages of enzymatic action and hydrolysis probably to more highly reactive forms and is finally oxidized into water and carbon dioxide.

There is still another influence or reaction which plays what may be considered in some respects the most important part in the metabolism of carbohydrates, and this is the internal secretion of the pancreas,

usually called insulin. When the formation of this hormone is interfered with or totally destroyed, diabetes mellitus develops. The nature of this hormone is not known. It is certain, however, that this substance is unstable to the effects of heat and digestive juices. It is very probable that in the near future this substance will be isolated, since recent workers claim to have obtained crystals of insulin. It is found in the islands of Langerhans and is then distributed by the blood stream to the tissues of the body to facilitate carbohydrate metabolism. Apparently insulin affects the sugar molecule, changing it to a more highly reactive form which is readily oxidized. The theory that insulin starts the proper oxidation of carbohydrate in the tissues, thus acting in effect as the "spark," receives considerable support. That insulin is essential to carbohydrate metabolism is certain, but the manner in which it functions, like many other body functions, is far from understood.

It must be emphasized that the proper combustion of fat depends upon the normal combustion of carbohydrate. This is emphasized in diabetes when we find acetone bodies are formed from fat due to abnormal metabolism. A large amount of evidence is at hand which proves the fact and need not be considered here. The process involves "the transformation of carbohydrate into fatty radicles" and "the reduction of hydroxyl groups and condensations to form the long chains of the higher fatty acids."

The whole process of metabolism of the carbohydrates, as energy producers, as protein spacers, and as promoters of fat deposits, is one of the most important considerations in the study of nutrition and development, especially in cases of malnutrition where the feeding must be conducted with consideration for the caloric intake and its relation to body rest.

**Protein.**—The digestion and metabolism of protein has been studied for a much longer time than has that of the other food elements, and in consequence of this fact and other circumstances, the metabolic changes of this element in the human body are better understood. The enzymes through whose influence protein is digested are: pepsin, which is secreted as pepsinogen and is activated by hydrochloric acid into pepsin and in turn changes protein, through a process of hydrolysis, into proteoses and peptones. It has been doubted that rennin exists as such and apart from pepsin and, in fact, they have never been separated. Evidence, however, points to its being a separate substance. Its function is to curdle milk and in this way prepare it for further digestion by rendering the casein insoluble. It acts in a very weakly acid medium so

that when the hydrogen-ion concentration (relative acidity) increases, the action of rennin is checked until it passes on to a more congenial medium. Hydrochloric acid, though not an enzyme but a mineral acid, is an important factor in the gastric digestion of proteins. Its known functions include the changing of pepsinogen into pepsin, of protein into metaprotein, the acidifying of the chyme to an extent suitable to the action of pepsin, which requires a relatively high acidity for its action, and possibly further participates in gastric digestion, particularly in the production of intermediary substances. Passing from the stomach to the intestines we find trypsin, or its proferment trypsinogen, a product of the pancreatic secretion. This ferment has an action which practically duplicates the pepsin of the stomach in that it changes proteins to proteoses and peptones, but goes further in splitting the proteins into polypeptids and amino-acids. Erepsin is a product of the intestinal secretions and attacks the peptones already formed, changing them to amino-acids. Again, we find rennin as a product of the pancreas and intestinal secretions. Other substances than the active ferments are found in the intestinal secretion. These substances, although playing no active rôle in the digestion of protein, serve an important function in activating the enzymes which either rely on them for this stimulation or actually stimulate certain glands to secrete.

Enterokinase functions as an activator to the pancreatic secretion trypsin, which is secreted as the proferment trypsinogen and is changed by the action of this activator into trypsin. Secretin produced by the glands of the intestines is believed to actually stimulate the pancreas to secretion. However, the pancreas is not altogether dependent on this hormone for its activation; evidence is not lacking which tends to prove that this organ may be stimulated to secrete through the action of certain mineral salts, particularly calcium and magnesium.

Besides these briefly described enzymes, there are certain enzymes scattered through the tissues of the body which play important parts in the metabolism of the absorbed products of intestinal digestion. Among these are a certain group of autolytic enzymes which perform the very important function of splitting protein into nitrogenous bases and amino bodies. A number of other enzymes taking part in the more intricate steps of intermediary metabolism, are imperfectly understood, and call for no other mention in this work.

The protein on ingestion passes through the mouth without being acted on, since no proteolytic ferment exists there, and on reaching the stomach is converted into metaprotein by the hydrochloric acid and



into proteoses and peptones by the pepsin. When the intestine is reached this process is continued; that portion of the protein which has escaped the proteolytic action of the gastric secretions is acted on by the trypsin and formed into proteoses and peptones, polypeptids and amino-acids, while that portion which has been acted on by the gastric digestion, together with the proteoses and peptones formed by the trypsin after reaching the intestine is further split into polypeptids and amino-acids. The peptones are in turn acted on by the erepsin to form amino-acids. After this final change, it is likely that the absorption occurs as amino-acids. Absorption takes place through the vessels of the intestinal villi, although under certain exceptional conditions some absorption may occur through the lymphatics. It is then carried by way of the portal circulation to the liver, where the greater portion of amino-acids are deaminized. A certain amount of amino-acids is conveyed by the circulation to the various tissues of the body, where certain ones are absorbed at this time directly by the tissues through a process of selection. There they remain until drawn upon for the purpose of tissue repair or tissue building and are available at all times. The various processes of metabolism of protein are intricate and between those stated occur many intermediary products caused by a large number of enzymes whose action is only too imperfectly understood as yet. Whatever portion of protein is not used in tissue building and for the purposes of growth is employed by the body for energy.

**Mineral Salts.**—Until quite recent years little or nothing was known of the metabolism of mineral salts; nor had much attention been paid to them. It is true that suspicion rested on some of them as vaguely connected with certain phenomena of deranged nutrition and other abnormal states, but to a large extent they were ignored principally because nothing was known about them. Although a great deal has recently been added to our knowledge of these materials and their importance in the processes of digestion and metabolism, particularly in relation to growth and development, only a relatively small amount of actually proven fact is as yet possessed. Attention has been drawn to certain of the inorganic elements and intensive study made of them because of the increased interest in rickets. That these salts play important parts is certain, not only in the normal functions of the body, but also disturbance of their equilibrium is a potent factor in a number of abnormal states. About fifteen inorganic elements are known to enter into the composition of the human tissues, of which



nine or ten are classed as mineral. Only a few of the more important of these will be considered here.

Mineral salts enter the body either as unattached salts (such as sodium chlorid as a condiment) or in solution or contained in the animal and vegetable food of daily consumption. They pass through the mouth unaffected, and are not absorbed in the stomach. They are absorbed entirely from the intestines and are excreted mostly from the large intestine, but to a lesser extent also through other secretions and excretions. After absorption they enter into the tissues through complex processes, not yet well understood, although a number are merely in solution in the tissue juices.

The more important mineral salts are those of calcium, magnesium, sodium, phosphorus, chlorin, and iron.

*Calcium Salts.*—Calcium is absorbed entirely by the intestines. Its excretion is almost entirely from the large intestine. Since calcium phosphate is practically insoluble its absorption is slight if indeed any occurs. Calcium salts are not absorbed. The calcium content of the blood remains at least during health at an approximately definite level. In children this is about 10 to 11 milligrams per 100 cubic centimeters. The degree of irritability of both muscles and nerves is markedly affected by calcium and this phenomenon has an important bearing on several conditions, notably in the case of tetany. A definite amount of intake is necessary to insure an adequate absorption and to maintain the balance between intake and output. The relation between the intake of calcium and fat must be a definite one to insure adequate calcium retention. Cod-liver oil definitely influences calcium absorption and retention. Whether this is due to the oil or to the antirachitic vitamin D, we are not prepared to state at this time. There are a number of intermediary processes in the retention of calcium which are as yet not understood. In the case of rickets, the calcium retention is disturbed and this process is intimately associated with the intake, absorption, and metabolism of phosphorus. The administration of calcium chlorid alone does not increase the retention of calcium except for short periods, without the simultaneous administration of vitamin D, conveyed best in cod-liver oil. The blood content of calcium cannot be increased beyond normal through administration alone (parathyroid hormone). Again it may be said that the metabolism of calcium is intimately associated with phosphorus. The principal utilization of calcium in the body is in bone formation. This salt is in larger amounts in the human body than is any other of the mineral substances, and

about 99 per cent of this may be found in bone. Even though the calcium of bone is in quite stable combination it may be drawn upon when needed by other tissues of the body when their normal supply is lowered. This is particularly true of a pregnant woman who needs a larger amount of this salt than is needed under any other condition, for the purpose of supplying an adequate amount needed for the rapid utilization in bone formation by the fetus. When a pregnant woman's diet is insufficient in calcium to meet this requirement the calcium stored as a part of the bony skeleton is drawn upon to meet this need. The average American diet, especially in cities, is signally deficient in calcium and this deficiency is more marked in this substance than is the case with any other mineral. This is, of course, of vast importance during the period of growth, at which time the human body needs calcium more than at any other period of its existence. Although cow's milk contains considerably more calcium than does human milk, the infant absorbs more calcium from the latter than from the former, or at least absorbs it more readily.

*Magnesium Salts.*—The metabolism of magnesium is closely related to that of calcium. The absorption of magnesium is entirely by the small intestine and its excretion almost entirely from the bowel, except that a small amount is excreted through the kidneys. The blood content of this salt is not fixed or even fairly constant. It varies from 1 to 5 milligrams per 100 cubic centimeters, according to varying circumstances. Little is known of its metabolism after absorption, nothing being known concerning the intermediary steps, but it is well recognized that it has a markedly depressing effect on the nervous system.

*Sodium Salts.*—Sodium is absorbed from the intestines in the form of chlorids and carbonates. Its excretion is largely from the urine, and in this respect differs from the other mineral elements. Sodium circulates as bicarbonate and in this way takes part in the maintenance of the alkali balance. The amount is not fixed but varies even in health from 50 to 70 volumes per cent. Environmental influences cause variations even beyond this limit. The amount of circulating sodium, and indirectly the amount of intake, influences to a large extent the retention of fluid and consequently the fluid balance of the body. Subcutaneous injections of normal saline solution increase the total fluid of the body and this increase is maintained for a longer period in wasting diseases and conditions than in normal children. The administration of sodium does not affect nervous excitability, so far as we know, in

any condition except in spasmophilia. This is well recognized clinically, but the manner in which such action is produced is not known. That it promotes the excretion of calcium has been advanced as one theory, while another theory suggests that it counteracts the normal depressing action of magnesium. Excessive administration of sodium may produce an alkalosis, with attendant clinical manifestations.

*Phosphorus Salts.*—There is apparently no relation between the amount of phosphorus in the food consumed and its absorption. That phosphorus is important in the formation of body protein is certain; also certain intermediary substances in the formation of fat within the body contain this element. The importance of phosphorus in bone formation is too well known to call for more than passing mention. Fat has been considered an influence in promoting the absorption of phosphorus. This idea arose in all probability through the administration of cod-liver oil and it is probable that this influence should be attributed to vitamin D. It is doubtful if any influence in this direction exists in the normal child.

*Chlorin Salts.*—The importance of this element depends largely on the fact that a number of the important salts are utilized as chlorids and the rôle it plays in the formation of hydrochloric acid.

*Iron Salts.*—Iron is one of the important and essential inorganic substances of the human body. It is ingested as organic compounds contained in a number of foods in daily use. Iron is absorbed by the intestines and conveyed to the circulation by means of the lymph channels, and is stored, after assimilation, in the liver, spleen, and bone marrow. Its elimination is by way of the intestine. That hemoglobin is dependent on iron is well known but how the body builds this up from the material supplied is not known. The utilization of inorganic iron in hemoglobin formation is a much debated subject, but is doubted by a majority of observers. There is some evidence to show that stimulation to blood corpuscular formation is effected by inorganic iron. One of the most important phases of iron metabolism is the fact that human milk contains a larger amount of iron than cow's milk. Also that even human milk contains an amount insufficient to meet the needs of the infant, the latter drawing on its reserve, which is large at birth, to make up the deficiency. When cow's milk is given, the supply is far more inadequate, and this is usually at a time when the reserve has been drawn on heavily. Therefore, too long continuance of milk feeding produces the well-recognized "milk anemia" of infancy.

## CHAPTER IV

### PRENATAL GROWTH AND NUTRITION

Having considered the various food elements on which the growth and development of the organism depend, the chemistry of their digestion, and the various metabolic changes through which they pass in the process of tissue building, we begin the study of the phenomena resulting from these processes. In order to form a proper estimate of the interrelation of growth, development, and nutrition, we must go back to the moment of the fructification of the ovum in utero. By the union of the female germ and the male sperm a new life is created, a new being is started on its career. Biologically, this fertilized ovum is an adult human being in embryo, and during the first few weeks of its existence it is difficult, if not impossible, to determine to what species it belongs. It is possessed, however, of every possibility of physical characteristic, as well as of every mental and moral attribute with which the adult product may be endowed. The color of the eyes and hair, the shape of the head, ears, nose, hands, and feet, and within certain limits the stature, mental attainments, personal characteristics, and even the standard of morals of the individual are ordained at the eventful moment of fructification. These matters are determined by cell factors, transmitted to the individual by the parents and through them by the ancestors. This we have learned by the study of biological processes, and it has been shown that the combination and order of transmission of cell factors is not haphazard nor by chance, but is definitely governed by and strictly in accordance with the well-known Mendelian law, the only chance entering into the process being the chance meeting of two individuals who eventually mate and reproduce their kind, that is, the bringing together of the cell factors of the ancestors which are united in each of the two mating individuals.

It will be noted that it was stated "within certain limits the stature, mental attainments, personal and physical characteristics, and even morals were determined by cell factors." Biologically, this statement is correct, but must be modified to the extent that environment must be reckoned with. Stature and the process of development are modified by nutritional influences and pathological processes, both antenatal and



postnatal; while other environmental influences (education and contact with individuals) may influence the mental attainments as well as the moral character of the individual. And yet, whatever level the individual may attain, even when influenced by nutritional, educational, or personal environment, is limited by the possibilities (cell factors) bequeathed at the moment of conception. As has been shown in a previous chapter, a race relatively short of stature, may, under the influence of better food conditions, provided these conditions are sufficiently prolonged, actually increase in stature. Whether this is due solely to better food or also to the activating influence of certain food factors on glands supposed to influence stature, is not as yet determined. A child endowed with a certain level of mental efficiency reaches a capacity level which is largely determined by the educational advantages enjoyed, or develops a moral standard through the influence of those with whom it comes in contact. But we must never lose sight of the fact that the height of the level of mental efficiency is limited and the ability to acquire information or a high moral standard, or to apply knowledge, or initiate conduct, is inherent. Therefore, however much we may extol these accessory environmental advantages, the possession of cell factors of a type which render such development possible, must be presupposed to exist at the moment of conception.

But let us return to a consideration of the fetus, and study its growth from conception to birth. Exact data of length and weight at various periods of gestation are hard to secure and in the observations made by different authorities, the variations are so marked that we must take the evidence of a single observer or average all figures. It is sufficient for our purpose to make a few comparisons based on approximate figures. At present, our best criterion of growth and development is the ratio between length (stem or body) and weight at various ages, though certainly at full term, length is a better criterion than weight by which to judge the age of the fetus.

The rate of growth of the human ovum is one of the marvels of nature. The mature human ovum weighs 0.000004 of a gram, and at the fourth week of fructification weighs 0.04 of a gram, an increase of one million per cent. By the end of the second week the ovum has attained the length of 4.2 millimeters. From this time on, the rate of growth is relatively much slower but still rapid enough to be marvelous. The measurements and weights may best be expressed by the following table:



Age	Length	Sitting Length, Centimeters	Weight, Grams *	Living Weight, Grams *
By end of				
1st month ....	7.5-10 mm.	....	1.4	....
2nd month ....	3 cm.	....	3.9	....
3rd month ....	8.4 cm.	6.1	14.2	....
4th month ....	10-15.8 cm.	11.6	87	....
5th month ....	18-22.5 cm.	16.4	261	....
6th month ....	30.3 cm.	20.8	552	....
7th month ....	36.1 cm.	24.7	971	3469
8th month ....	41.4 cm.	28.3	1519	1190
9th month ....	46.2 cm.	32.1	2200	1738
10th month ....	50.5 cm.	....	3250	2419

\* Scammon and Calkins, *Proceedings of the Society for Experimental Biology and Medicine*, 1924, 21: 549-51.

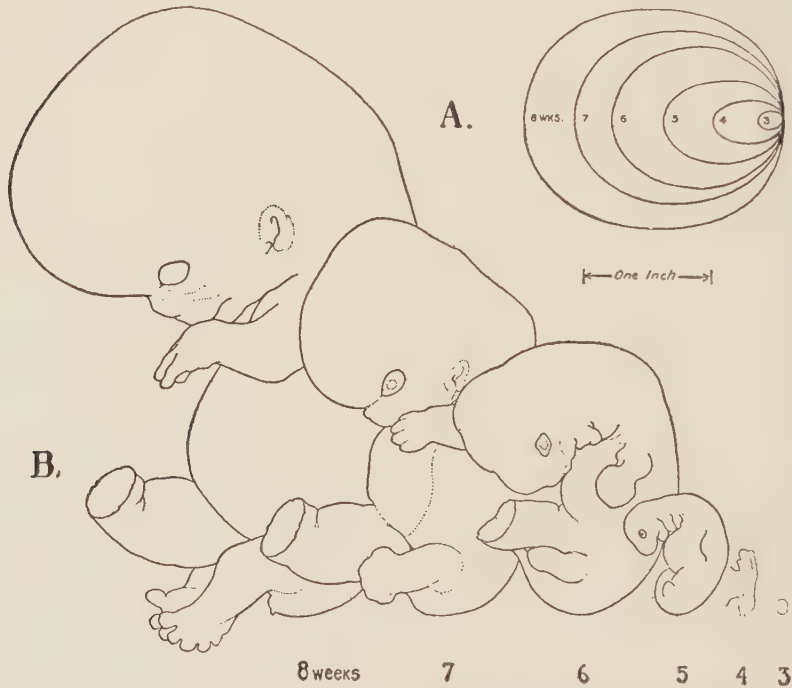


FIG. 1.—SIZE OF OVA AND EMBRYOS FROM THREE TO EIGHT WEEKS. A. shows the average natural size of human ova from three to eight weeks old. The villi are included in the outlines. B, human embryos from the third to the eighth week of pregnancy, enlarged five diameters. Reduced one-third in reproduction. (From Jordan and Kindred.)

The weights here recorded through the sixth lunar month are dead weights. The weights recorded for the seventh, eighth, ninth, and tenth months are also dead weights. In parenthesis will be found the living weights which are the sum of the dead weights plus 219 grams.

After the second month, the rate of increase per month is as follows: 74, 11, 1.75, 0.82, 0.67, 0.5, 0.47, and 0.45. In the same period of time, the length of the fetus increases from an approximate 4.2 millimeters to 50 centimeters. These figures enable us to visualize the growth and development of the body as a whole during intra-uterine life. During the same period the various viscera develop both actually and in relation to the total body growth to a remarkable extent. The liver attains a weight of 135 grams (the average weight at birth), while the lungs of a full-term normal infant weigh 50 grams. The question of the difference between the weight of dilated or nondilated lungs (depending on whether death occurred before or after breathing) does not enter, because practically there is no difference, since, if a slight difference occurred, it is matched by the great variability of weight in different subjects. The combined weight of the kidneys averages between 20 and 25 grams, while the heart weighs about the same as the two kidneys. The brain in a full-term Caucasian child weighs approximately 350 grams. During intra-uterine life, the small intestine acquires a length of 340 centimeters, while the large intestine reaches in the same time a length of 66 centimeters. The gradual formation of bone reaches its climax of intra-uterine development so that at birth the total bony and cartilaginous weight is 15 to 20 per cent of the total body weight of a full-term normal infant.

The size of the full-term child varies considerably within normal limits and there are a number of factors which enter into the determination of the weight of the newborn infant. Practically all figures quoted are based on the law of averages which, in this instance, takes into consideration the duration of pregnancy as ten lunar months. It is well known that many pregnancies extend beyond this time and as a result, the child continuing to grow, during each day of its intra-uterine life is larger than the average at birth, directly in proportion to the length of time the pregnancy extends beyond the end of the tenth lunar month. The age of the mother at the time of pregnancy has also an influence on the weight of the offspring. For example, several successive pregnancies between the ages of twenty-five and thirty-five will probably show an average-weight infant for the first of the series and a gradually increasing weight of each succeeding child up to the mother's thirty-fifth year, after which each succeeding child would be smaller than the product of the preceding pregnancy. The quoted figures apply to the Caucasian race. Racial differences in the weight of newborn children are well recognized outside of the Cauca-

sian race, but there are noticeable differences between the children of the various nationalities which comprise the Caucasian race.

One observation on racial differences is of very practical importance to us in America and particularly to those living in the southern states. That is that Negro infants are notably smaller at birth than white infants. This fact to some extent explains why the incidence of stillbirths among Negroes is not larger than it actually is, since a very large number of Negro women have contracted pelves because of the large amount of rickets in this race. Were the Negro children of the same average weight at birth as white children, there could be little doubt that the incidence of stillbirth would be higher. Offspring of the well-to-do, or leisure classes, who, as a rule, are also well nourished, are usually larger than the average. This observation likewise applies to representatives of the various strata of society, the offspring of women of the upper strata being noticeably larger than those lower down in the scale. Observations which tend to show the influence of social and economic levels are borne out very largely by the fact that women from the poorer walks of life who reside in a maternity hospital for a reasonable period before delivery (a month or more) give birth to infants who are measurably larger than those from the same environment who enter the hospital just before the birth of their child, or who are confined in their homes. This shows the influence of improved nutrition during the last weeks of pregnancy.

**Fetal Nutrition.**—The much-debated question of whether the unborn child is a part of its mother or is merely a parasite, calls for no discussion at this time. Suffice it to say that the nutriment of the fetus is derived entirely from the blood of the mother, the placenta elaborating from the mother's blood the food necessary to nourish and promote the development of the unborn child.<sup>1</sup> The actual separation of materials from the mother's blood which are suitable for or needed by the fetus for purposes of metabolism and development is performed by the chorionic villi which possess the power of selective action. This is accomplished by "transforming non-diffusible substances into diffusible substances, thus rendering them transmissible to the fetal circulation." Thus it will be seen that the food acquired by the infant's body has been ingested by the mother, subjected to the various processes of digestion and after more or less complete digestion ab-

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<sup>1</sup> A fairly comprehensive account of the phenomena incident to maternal and fetal nutrition may be found in *Physiologie Obstétricale Normale et Pathologique*—H. Vignes.

sorbed by her circulation, transmitted through the same circulation *directly to the circulation of the fetus*, to be metabolized by the fetus through the action of its own tissues and organs. In this way, the fetus itself generates a large amount of energy.

It is interesting to note that the amount of iron and the calcium and potassium salts received by the fetus from the maternal blood stream is not constant through the whole of pregnancy, but varies, according to the needs of the fetus, with different periods of pregnancy. This phenomenon has been attributed to a selective action on the part of the placenta.

During the whole period of gestation, there is a constant adjustment of the tissues and organs of the mother to meet the demands placed upon them by the development of the fetus. There is a hypertrophy, or at least a congestion, of many of her tissues and organs. Her spleen enlarges, and the volume of her red bone marrow increases, while the white marrow shows no demonstrable increase. This is obviously to care for the increase in the hematopoietic function. Certain of her ductless glands are increased in size in the same manner. The resources of her body, in stored material, are constantly being drawn on, varying according to the need of the particular period of development of the fetus. This is especially true of certain of the mineral salts and notably so of calcium. Generally speaking, the average balanced ration in daily use supplies a sufficient amount of materials needed to meet the additional draft, but if this balance is not maintained, the mother must literally give of herself in order that her unborn offspring may be supplied with an adequate amount of elementary material. Observations made on lower animals and fowls have demonstrated this in an interesting fashion. A hen, during the laying period, was producing eggs with normally hard shells. During this period and the subsequent one of experimentation, she was kept on a measured diet, including a fixed amount of lime or lime-producing food. Her leg was then broken and during the process of callous formation incident to the healing of the bone, she produced eggs having no hard shell, showing that her reserve of calcium was not sufficient to meet the demands of egg-shell formation and bone regeneration at the same time without an increase in the supply of bone-forming material in her food. It will be seen by all of these preceding observations that the production of offspring is essentially one of self-sacrifice on the part of the mother.

The law of compensation comes to her assistance, however, in most instances. Her appetite increases during gestation in direct proportion



to the age of her unborn offspring. An observation has been made by one investigator (Bar) on a woman who required during her gestation 16 grams of nitrogen in her diet, and four months afterwards, while not nursing her child, took a diet containing only 12 grams of nitrogen. Observations were then made on the retention of nitrogen by taking a balance of the food ingested and the food excreted, and it was shown that in the woman studied there was first a phase of retention of nitrogen and then after saturation, a phase of excretion, which was increased beyond that of normal nonpregnant women, while finally during the second half of pregnancy there was a retention proportional to the needs of the infant. It was further shown that a large part of the retained nitrogen is used by the fetus and its attachments, in accordance with figures published by various authors, stating the needs for amounts of nitrogen in various ages of fetal life. It is still further shown that the requirement of the fetus for nitrogen is at its maximum just when the mother retains the most nitrogen.

Under normal conditions the mother gives the proper protein molecules to the fetus and in order to face this necessity she stores protein within herself. Towards the end of pregnancy the human fetus has need of about 1 gram of nitrogen per day. The mother retains from 2 to 4 grams. The excess of nitrogen stored is not found in the mother's blood and it is not known in what form it is stored nor in which organ. The retention of nitrogen is favored by the retention of phosphorus. After delivery a partial loss of the nitrogen is again observed and this is largely in the form of urinary nitrogen.

Observers have also found in pregnant women a diminution in urinary carbon which indicates its retention. This substance serves in part for the nutrition of the fetus and plays a part in oxidation, since the carbon dioxid exhaled is greater and the calories are greater. A further use is found for the carbon as an aid to the storage of neutral fats which accumulate in the cellular tissue of the fetus. It has been shown that the excretion of urinary phosphorus is less in pregnant women than in normal women, as a rule. In some cases the excretion is about the same and occasionally it is more than in normal women, but this is distinctly an abnormal state and is probably due to an increase in the ingestion of this substance. In women on an optimum diet, retention of phosphorus is invariably the case. A greater portion of the retained phosphorus goes to the fetus and its attachments. The metabolism and fixation of phosphorus by the fetus is slow in the early months but increases rapidly toward the end of gestation. During the



period of gestation the mother also shows a retention of chlorin and sulphur. With regard to iron, the mother furnishes the fetus a sufficient amount for it to form hemoglobin and also to establish a reserve within the fetal body. A part of this the mother takes in her daily diet but a considerable amount may come from her own reserve supply. If the tax on her iron reserve is large, an impoverishment may occur which frequently leads to a severe state of anemia. The need for lime in fetal development is naturally very great and this need increases with the advance of fetal life, being relatively very small in the early months. The skeleton of the newly born infant is less rich in lime than older children or adults, but when this is compared with the large water content of the body in early intra-uterine life, it will be found that the lime content is relatively large. The nervous system is particularly rich in lime. This lime content is subject to many variations in infancy, as is well known, frequently causing a marked imbalance in nerve excitability. It is quite likely that the same thing may occur in early intra-uterine life. A close analogy exists in this particular between nervous excitability and muscle contractility. In the later months of gestation the demand of the fetus on the mother in lime salts is particularly severe. It has already been intimated that it is hardly likely that the fetus takes its total calcium requirement from the daily intake of food by the mother; consequently her reserve supply is drawn on. It is stated, not altogether without evidence, that the mother's temperament plays an important part in the absorption and retention of various salts.

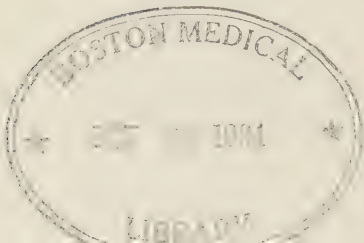
It is quite evident that a study of fetal metabolism is largely that of the pregnant woman. It is supposed that after the fetus receives into its blood stream the food from the mother's blood, the function of adaptation and synthesis is performed by the fetal tissues; however, it is not at all unlikely that a considerable amount of this metabolic process has been performed by the mother herself and that a large portion of fetal activity (functional) is merely an absorptive process.

## CHAPTER V

### DEVELOPMENT

Child life is generally divided into five periods for purposes of sociological study. These periods are antenatal, neonatal, infancy, pre-school or runabout, and school ages. The antenatal period is the only one to which a definite time limit can be assigned. The neonatal period, or the period of the newborn, is a variable one, depending on the custom of an individual locality. By some it is regarded as ending at the second week, by others at the fourth week, but since there are certain characteristics of the newly born which endure for four weeks, it is probably wiser, or at least more convenient, to consider the first four weeks of independent life as the neonatal period. The term infancy, likewise, is applied to a period with wide, arbitrary variations. When it was the custom for infants to be kept on mother's milk or some form of cow's milk exclusively for twelve months, these months might have been regarded as the period of infancy. Based on this criterion, infancy should end now at the sixth month since it is an almost universal custom to begin feeding semisolid food at this age. It would appear, however, to be more logical for us to determine such a point by independence of conduct and end this period when the child begins to walk. As a matter of fact, convenience and custom both suggest that infancy extends through the first two years of postnatal life, and in fact this is the prevailing custom sociologically and medically.

The next period is the preschool period, or the age of the runabout. It is in many respects the most dangerous period of child life, because the child is acquiring a definite independence of conduct and is not as constantly under vigilant supervision. It is during this period that many physical defects are acquired and a state of malnutrition develops which is insidious, except in those cases which are definitely continuations of a similar state in early life. This period ends more or less abruptly at the entrance of the child in school, which is anywhere from the sixth to the seventh year of life. From now on to puberty, we apply the term "school child" or "child of school age," obviously a remnant of a time when most individuals attended school only during these years. At least to the latter part of this period the term "youth" would be



better applied, because of certain physiological phenomena immediately preceding pubescence.

These periods are recognized solely for convenience and bear only a slight relation to anatomical and physiological development and growth. In the discussion which follows, developmental phenomena will be seen to occur in relation to different parts of the anatomy and to different tissues, rather than in definite time cycles. There are, however, cycles of growth in which individual tissues participate but with a distinct alternation.

Development, growth, and nutrition are terms which are often confused; in fact, it is difficult to differentiate sharply among them, because the underlying processes are both coöperative and interdependent. Broadly speaking, however, development may be defined as the evolution of the mature organism from the fructified ovum with a description of the processes taking place in the course of this evolution. Growth refers more directly to increase in stature, while nutrition implies the relationship between weight and stature; underlying both of which are the processes of food absorption and their subsequent metabolism.

It is necessary to observe briefly the development of various parts of the anatomy, certainly those grosser structures whose development bears a definite relation to growth or at least progresses at a rate which is more or less parallel with that of the body as a whole. Since increase in stature practically means increase in the skeleton, we will begin our considerations with the development of the osseous system.

**Various Parts of the Anatomy.—Bones.**—In the newly born and the young child, the larger proportion of bone consists of cartilage and fibrous tissue. As development proceeds, the character of bone changes and gradually takes on a certain density and hardness, so that in mature bone two-thirds of its composition is mineral, or inorganic material, and only one-third organic. By the end of the second year, ossification has advanced to the stage where the fetal character of bone is practically lost, but not until the twelfth year does bone reach the adult state. The growth of bone starts and proceeds from centers of ossification. Some of these originate in cartilage and some in membrane but in most instances from cartilage. There are in all, in the human body, approximately eight hundred centers of ossification; less than half of this number start before birth, while more than half do not appear until after birth. Of course, these centers of ossification do not represent individual bones in every instance; a number of them fusing so that

frequently a single bone is formed from several centers. In long bones, for example, the ossification center of the diaphysis appears first and progresses to a well-advanced state of bone formation before the center in the epiphysis develops at all. The number of centers of ossification and of separate bones varies at different periods of life. At birth, there are 270 separate bones and this number diminishes considerably during the first three years of life. Then there is a gradual increase, not steady, but varying in number from time to time, up to puberty (approximately fourteen years) at which time, approximately 350 masses, which may be identified as separate bones, are found. After this time, fusion takes place to an extent which constantly diminishes the number until the early part of the fourth decade of life, at which time the number of separate bones in the body is 206. There is great variation in the time of appearance of centers of ossification as well as the rapidity of bone development from these centers in different parts of the body. The time of appearance of centers of ossification and the rate of bony formation is not necessarily fixed by the age of the individual since a number of factors, other than pathologic ones, enter into the phenomenon. The influence of sex is striking, while the process varies appreciably according to the number of pregnancies of the mother. There are in addition, striking variations which can be accounted for only through the influence of heritable traits. At birth, the bony and cartilaginous substances furnish 15 to 20 per cent of the total body weight and this proportion remains practically constant throughout life. The total increase of the skeleton as that of the whole body from birth to maturity is approximately twenty times.

*Skull.*—At birth, the circumference of the head, based on an average of a large number of individual measurements, and the average between the sexes, is 13.71 inches (35 cm.). The shape of the head is usually slightly ovoid, though it is often practically round. The size of the head at birth is vastly out of proportion to the other body measurements as compared with adult standards; in fact, its size is proportionately twice as large as that of the adult head. The portion of the skull which represents the brain cavity, or calvarium, is disproportionately preponderant over the facial portion. This disproportion changes only when the teeth erupt and the facial bones grow in the process of development of bony structure, and with the filling of the dental arches with teeth. During the period of the bony development of the facial portion of the skull, the various sinuses are formed, their development



also effecting much of the change of contour. Not until all teeth are fully erupted, usually about the fifteenth year of age, with the exception of the wisdom teeth, does the facial contour reach the adult type and the permanent proportion between the facial and the calvarial portion of the skull becomes established.

The growth of the head is most rapid during the first year of life, increasing during this period 4 inches (10 cm.). The rate of growth during this period, however, is not uniform, since it is far more rapid during the first half of the year than it is during the second half. During the second year, the rate of growth is far less rapid than during the first year, the circumference increasing only 1 inch (2.5 cm.) during the whole year. The rate of

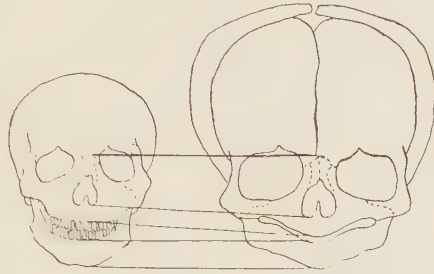


FIG. 2.—SKULL OF THE NEWBORN AND ADULT.

Drawn to the same face height to illustrate the relative proportions of the facial and neural skeleton at birth and in maturity. (After Holl. From Morris' *Anatomy*.)

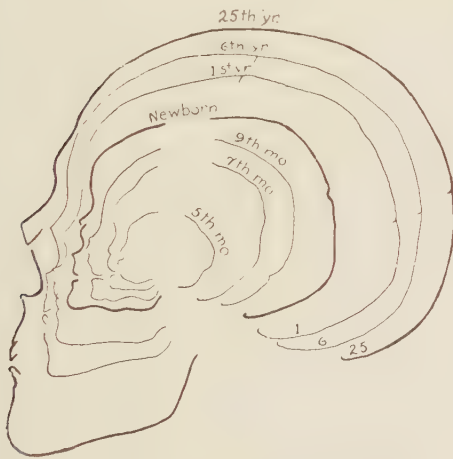


FIG. 3.—TRACINGS OF MEDIAN SAGITTAL SECTIONS OF THE SKULL AT DIFFERENT AGES, ILLUSTRATING THE RATE OF GROWTH OF THE CRANIUM.

(Based on the figures of Corrado and Welcker. From Morris' *Anatomy*.)

growth is still less rapid for the next three years, during which period there is an increase of only  $1\frac{1}{2}$  inches (4 cm.) in circumference. From then on, the growth of the skull is markedly slower, the circumference growing at the rate of only  $\frac{1}{2}$  inch in each five years until the full adult size is reached, usually in the early part of the third decade of life. After the fifth year the rate of growth is not steady but fluctuates between periods of relatively slow and rapid growth.

The size of the cranial vault, that is, the capacity of the calvarium, is of interest, since it is not only a manifestation of the growth of the skull as a whole, but because it also furnishes a fair estimate of the size and growth of the brain. At birth the capacity of the cranial

vault is about 400 c.c. and increases continuously, though not steadily, to 1,300 to 1,500 c.c., between which two figures the capacity of the average adult brain lies. The most rapid period of growth of the cranium is during the first two years, and in fact most of the growth takes place during this period. The growth periods from birth to adult life may be roughly divided as follows: the period of rapid growth from birth to seven years, the period of slow growth from seven to twelve years, and a period of slight acceleration of growth from then to maturity.

The sutures of the skull are ossified between the sixth and the ninth month; while the time of closure of the two main fontanels varies considerably even within normal (average) limits. The posterior fontanel is usually closed by the end of the second month. The anterior one closes anywhere from the sixteenth to the twenty-second month but averages eighteen months.

It has been stated that the development of the sinuses materially affects the contour of the facial portion of the skull. The time of development of the several sinuses is important, not only that we may have an appreciation of developmental processes occurring at various ages, but especially so since sinus infection in children is of recognized importance, and is often overlooked under the misapprehension that they are not sufficiently large to be considered when searching for foci of infection. It is quite probable that all of the sinuses develop earlier than it was once supposed and in exceptional instances attain considerable size when the individual is still quite young.<sup>1</sup>

The maxillary sinus, or antrum of Highmore, can be demonstrated "about the seventieth day of fetal life." In a full-term, normal infant, this sinus measures approximately  $7 \times 3 \times 4$  millimeters. By the seventh year, the measurements of this sinus are  $27 \times 17 \times 18$  millimeters. The growth of this sinus is rather uniform and the rapidity of growth is probably not influenced at all by primary dentition. By the fifteenth year, the sinus gradually begins to approach adult dimensions, which are  $34 \times 33 \times 23$  millimeters.

In the study of the frontal sinus, it has been shown that it does not always develop, during the embryologic stage, from the same place. In some instances it develops from the anterior ethmoidal cell, while in others it develops by direct extension of the frontal recess. Depending, therefore, on its developmental origin, the frontal sinus may or may

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<sup>1</sup> Size of mastoid cells from Kerrison; that of the paranasal sinuses from Schaeffer.

not be demonstrable at birth. As a rule, however, its existence cannot be proven until between the sixth and twelfth month of life, at which time each of the frontal sinuses measures on an average  $2 \times 2 \times 3\frac{1}{2}$  millimeters. From this point on they develop steadily and fairly rapidly, there being usually a measurable difference between the sizes of the right and left sinuses. By the fifth or sixth year the average size is  $7.5 \times 3.5 \times 6$  millimeters, increasing from this point quite rapidly to the fifteenth year, when they measure  $18 \times 19 \times 11$  millimeters. From this time on they rapidly attain the proportions as found in the adult skull.

The ethmoidal cells may be demonstrated quite early in fetal life and by birth measure  $5 \times 2 \times 3$  millimeters for the anterior group and  $5 \times 4 \times 2$  millimeters for the posterior group. Their size increases fairly rapidly, varying widely in different individuals and also in the number of cells composing the anterior and posterior groups. At five years they have attained a size of  $7.5 \times 6 \times 5.5$  millimeters for the anterior cells and  $7.5 \times 8.5 \times 6.5$  for the posterior cells, reaching an average size of 9 to  $16 \times 10 \times 5$  to 23 millimeters for the anterior and 9 to  $15 \times 14 \times 8$  to 20 millimeters for the posterior group by fourteen years of age.

The sphenoidal sinus is demonstrable about the middle of intra-uterine life and by full term reaches a measurement approximating  $2 \times 2 \times 1.5$  millimeters. The sphenoidal sinuses are quite inconstant in size and differ slightly as between the right and left. By the fifth year  $7 \times 6.5 \times 4.5$  millimeters may be taken as a safe average, while by the fourteenth year  $15 \times 10 \times 12$  millimeters is an approximate measurement.

The normal oval contour of the skull may be altered by various influences. In the early months of life, an oblique deformity sometimes occurs, causing temporary distortion of contour, though this rarely persists through life. Still more rarely, premature ossification of the sutures may result in permanent deformity. Syphilis often causes a premature closure of the fontanelles, while rickets delays the same process, sometimes beyond the third year. In achondroplasia, there is a failure of normal development of the tribasal bones of the skull; the brain at the same time growing at a normal rate, causes the upper part of the skull to grow out of proportion to the base, with a consequent disproportional increase in the size of the cranial vault.

As has been stated, the size of the skull is in a measure an index of the size of the brain. In hydrocephalus, the distention of the ven-

tricles increases the size of the cerebrum, with consequent delay in ossification of the sutures and closure of the fontanels with resulting disproportion of the size and shape of the skull. Likewise, in certain instances in which the brain fails to develop normally the closure of the sutures and fontanels is hastened with resulting diminution in skull capacity (microcephaly). A number of other varieties of deformity not well understood are often met with. Racial characteristics in the shape of the skull are well known and have been the subject of much study.

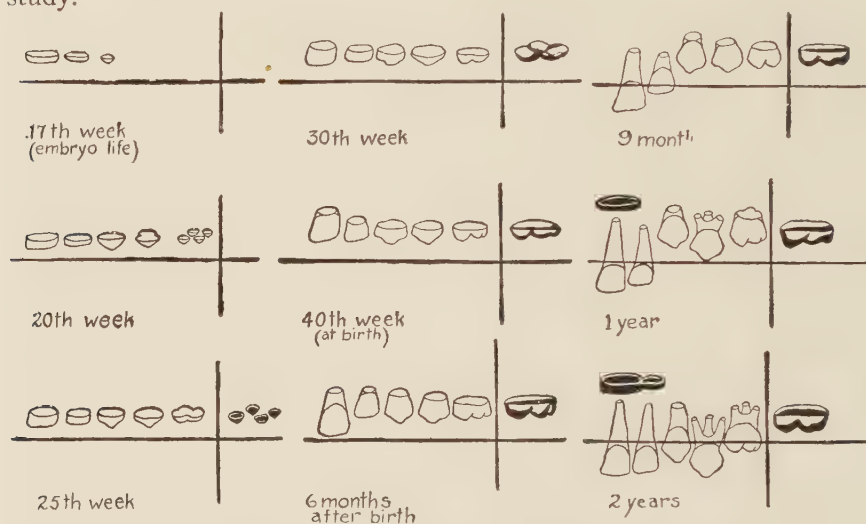


FIG. 4.—DEVELOPMENT, ERUPTION, AND ABSORPTION OF THE TEETH.  
(Designed by William J. Brady, D. D. S. From Grulee's *Infant Feeding*.)

The cycles of development of the head are quite definite. During the first few years of neonatal life there is a rapid increase in the size of the whole head due mainly to the growth of the brain, teeth, and jaws, as we have already observed. A second period of rapid growth occurs between the sixth and seventh years when the second teeth are developing. The third and last very active period of the development of the head is from the eleventh to the fourteenth year when the bones of the skull and face are thickening, the teeth developing rapidly, and the sinuses expanding.

*Teeth.*—We have observed the effect of the development and eruption of the teeth on the contour of the facial portion of the skull; a brief description of these processes is necessary to a clear understanding of the changes incident to this period. The germs of the temporary or deciduous teeth, and one group of the permanent teeth, the first



molars, are present early in fetal life. There are two more or less definite periods of calcification. During the fourth and fifth months of fetal life the centers of ossification of the deciduous teeth develop, while during the first five years of postnatal life the development of the centers of most of the permanent teeth occurs. Two secondary periods of development are marked by the calcification of the first permanent molars, which occurs in the last month of fetal life and the third molars, or wisdom teeth, which does not occur until the eighth or ninth year.

The process of calcification differs somewhat in the deciduous and permanent teeth. At the time of eruption of deciduous teeth, calcification is well advanced but not complete, but in the case of the permanent teeth this process is quite complete at the time of eruption. Calcification begins in the crown and proceeds to the roots, and not until this is accomplished is there permanent attachment within the alveolar sockets. In the shedding of the temporary or deciduous teeth, a process of decalcification occurs, beginning at the apex and proceeding toward the crown; so that by the time it is shed, practically the whole fang has been absorbed.

The average eruption of teeth occurs in accordance with the following tables:

#### *Temporary Teeth*

Two lower central incisors.....	6 to 9 months
Four upper incisors.....	8 to 12 months
Two lower lateral incisors and four anterior molars.....	12 to 15 months
Four canines.....	18 to 24 months
Four posterior molars.....	24 to 30 months

At one year a child should have.....	6 teeth
At 1½ years a child should have.....	12 teeth
At 2 years a child should have.....	16 teeth
At 2½ years a child should have.....	20 teeth

#### *Permanent Teeth*

First molars .....	6 years
Incisors .....	7 to 8 years
Bicuspid .....	9 to 10 years
Canines .....	12 to 14 years
Second molars .....	12 to 15 years
Third molars .....	17 to 25 years

The figures quoted are based on the average of a large number of observations. A wide range of variation occurs in children who are apparently perfectly normal. Besides these variations, others occur, the most constant being those due to heritable traits. In some families there is a uniformly early eruption of teeth, while in others there is a correspondingly late appearance. Certain diseases also have a marked influence on the time of eruption of teeth. Syphilis causes early eruption (*c.f.*, early closure of fontanel), while rickets has been blamed for their late eruption. Since the intensive study of rickets during recent years, considerable doubt has arisen on this point. Feeble-mindedness has a definite influence in causing late eruption. Scurvy is also influential, not only in delayed eruption, but also in determining the character of the teeth.

The state of nutrition apparently has a marked influence, not only on the time of eruption of teeth, but on their development as well. It is not unlikely that certain metabolic derangements of the mother during the antenatal period, as well as intra-uterine disease in the fetus, may materially influence this whole process. The shape of the teeth may be altered by disease, such as syphilis (Hutchinson type), or by a state of malnutrition, which is supposed to cause certain notched teeth, other than those of the Hutchinson type, or through the transmission of heritable traits. Malocclusion, a condition which is both interesting and important, has many causes. Among these causes may be mentioned irregular eruption of teeth, which is usually a heritable trait, mouth-breathing (disputed by some), sucking of pacifiers, fingers, and tongue, improper methods of swallowing, and other habits bringing unusual pressure against the teeth. Most of these causes act before the bones of the alveolar processes have become completely ossified.

*Spine.*—In the development of the spine, only the free portion will be considered, the sacrum and coccyx being of no special interest. The growth of the spine is important in any study of child life, since it furnishes the criterion of growth of the torso in comparison with the extremities, an important consideration in all studies of the total body height. The increase of the length of the spinal column is brought about by the bony development of the vertebræ from two separate centers of ossification. Growth in childhood is mainly from the primary centers, while after puberty it takes place from the secondary centers, or those of the epiphyses.

The ossification of the spinal bodies does not proceed at a uniform rate, but rather in cycles of rapid and slower growth, as is usually the

case with other bones of the skeleton. An interesting fact is that some of the ossification begins in the cervical bodies and proceeds downward, while some starts in the lower portion of the spinal column and proceeds upwards.

During fetal life, the spinal column is a distinct arc with an anterior concavity. At birth this curve straightens out until the line of the spine is more nearly straight but still has a slight anterior concavity. As growth proceeds, the normal spinal curvatures gradually take shape, so that at six years the dorsal segment has an anterior concavity which nearly approximates the adult curve; the cervical segment having a perceptible anterior convexity, while the lumbar region is still nearly straight. Between the thirteenth and fifteenth years the spine shows less curving in the cervical and dorsal regions than at six years, while the lumbar spine has begun to show a marked anterior convexity. This is due to the

thickening of the anterior portion of the lumbar vertebræ and the intervertebral disks and is indicative of the tilting of the pelvis incident to puberty and is particularly marked in the female. The full curves of the spine alternating between the cervical, dorsal, lumbar and pelvic regions, characteristic of the adult, do not develop until full maturity.

At birth the length of the free vertebral spine is 8 inches (20 cm.) and is 40 per cent of the total body length. From this time to the end of the second year the increase in length is phenomenally rapid, reaching  $17\frac{3}{4}$  inches (45 cm.). From now on until puberty the growth is very slow, only 2 more inches (5 cm.) being added, bringing the total length of the spine at puberty to  $19\frac{3}{4}$  inches (50 cm.). From birth to adolescence the vertebræ more than double their size and this growth takes place at different rates according to three rather definite periods: "From birth to three years, during which from one-half to two-thirds of the entire postnatal increase occurs; second, from three to sixteen

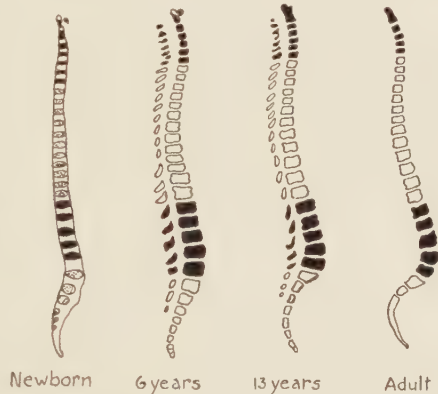


FIG. 5.—MEDIAN SAGITTAL SECTIONS OF THE VERTEBRAL COLUMN AT VARIOUS AGES, ILLUSTRATING THE DEVELOPMENT OF THE NORMAL SPINAL CURVATURES.

Cervical and lumbar vertebræ indicated in black. Based in part on the figures of Bardenheer, Williams and Cunningham. (From Morris' *Anatomy*.)

years, a period of little if any growth; third, from sixteen years to maturity, during which approximately one-third of the total growth takes place." (Scammon and Holt.)

*Extremities.*—The sequence of development of the bones of the extremities is more irregular than the bones of other portions of the skeleton. The shafts of all long bones show mature characteristics about the fifteenth year, but completed bones, *i.e.*, those in which all centers have fused with the main portion of the bone, do not present until well into the third decade. The clavicle presents the widest limits in periods of ossification. It is the first bone in the whole body to show ossification, while the last center of ossification does not appear until near the twentieth year, and this center is not finally united so as to form an integral part of the bone until about the twenty-fourth year.

The growth of the upper extremities is more rapid than the lower extremities during the first month of fetal life. With this exception, the growth of the lower extremities is far more rapid than that of the upper during the whole prenatal period and is especially disproportionate during the last month of intra-uterine life. The very rapid growth of all extremities during fetal life continues through the first year of post-natal life, after which there is a definite lull in the rate of growth but showing subsequent periods of acceleration at the fourth, eighth, and twelfth years. During all of this time, although both extremities are growing steadily, the rate of growth in the lower extremities is greater than that of the upper.

The bones of the extremities alternate in growth between length and breadth. They increase in diameter for a period of six months and in length for the next period of six months. These periods are opposite for the serial bones of the same extremity; that is, while one is growing in length the other is increasing in diameter. For example, while the femur is increasing in length, the tibia and fibula are increasing in diameter, and while these two bones are increasing in length, the femur is increasing in diameter.

The shaft of the humerus begins to ossify early in fetal life and is completely ossified at puberty. There are a number of epiphyses, however, representing the head, tuberosities, and condyles, which unite with the bone at varying intervals, commencing in early childhood and continuing up to the eighteenth year, at which age the bone may be considered complete. The humerus at birth is 7.7 centimeters in length



and averages 20 centimeters in the adult. The latter figure is subject to considerable individual variation.

In both the ulna and the radius ossification begins early in fetal life, in fact before the eighth week, and shows final union of all component parts, in the case of the ulna by the seventeenth year, and in the radius early in the third decade.

The ossification of the bones composing the carpus is entirely a postnatal process, terminating a little beyond puberty. The metacarpal bones and the phalanges are usually completed by the twentieth year.

The centers of ossification of the bones of the lower extremity are much later in making their appearance than are those of the upper extremity. In the case of the femur, the first center to develop, that of the inferior epiphysis, is present at birth, while the final union of ossification does not occur until early in the third decade. The union of epiphyses in this bone is in reverse order of their appearance.

Quite a marked change in the shape of the femur occurs from that of fetal life to that of maturity, more so in fact than in any other long bone of the skeleton. One of the most striking of these changes is seen in the fact that at birth the bone is practically straight, while at maturity there is marked anterior bowing. This change occurs when the muscular development incident to the assumption of the erect posture progresses. The length of the humerus at birth is 9.3 centimeters and 26 centimeters in the adult.

At birth the tibia shows one center of ossification, but the last union of the segments does not occur until well into the third decade. The fibula shows no ossification at birth and develops according to the individual, anywhere from the fifteenth month to the third year. Complete union does not occur until the twenty-fourth year.

For the purposes of our present study, relatively little importance attaches to the development of the bones of the hands and feet. The degree of ossification, however, of the carpal bones is of distinct significance in the diagnosis of certain conditions, accompanied by feeble-mindedness.

Peculiar interest attaches to the ossification of the bones entering into the formation of the knee-joint in determining the maturity of the newborn infant. In full-term normal infants the inferior femoral epiphysis can be demonstrated about the first part of the tenth (lunar) month of fetal life, while by the end of that month the upper tibial epiphysis is shown. Children born with both of these centers, weighing 2,700 grams, and measuring 47 centimeters or more,

are full term. Children with both of these centers, weighing 2,600 grams, and measuring 44 or more centimeters, are very nearly if not quite at term. The same approximation to term applies to those children weighing 2,800 grams and measuring 44 centimeters or more, even though a single center is present. The presence of both of these centers is evidence of eight and one-half calendar months of life; while all children without ossification centers and weighing less than 2,500 grams are certainly less than term.<sup>2</sup>

*Thorax.*—The ossification of the component structures of the thorax, other than the spine, are of relatively little importance for the purposes of this work, the shape and increase in size being of far more importance. There is no single part of the body which is a better index of development or the state of nutrition than the size and contour of the chest.

At birth the thorax may be described as a truncated cone, flaring widely at the bottom. It is nearly round, deviating from this shape to the extent of a slightly anteroposterior flattening. As a child grows, the contour gradually changes to an oval, which change is produced by an excess of the growth laterally over the growth anteroposteriorly. The relation of the anteroposterior diameter to the lateral, which gives the final shape of the adult chest, is not established until puberty or beyond. The growth of the chest vertically relative to the length of the trunk, takes place largely in the anterior wall, due to the growth of the sternum. This is due largely to the attachment of the ribs to the spine, which is the most important part of the posterior wall. At birth, the upper margin of the sternum is opposite the second dorsal vertebra, while the tip of the xiphoid cartilage is opposite the tenth dorsal vertebra. These landmarks closely approximate those of the adult chest, but during the period of growth there is a relatively greater lengthening of the sternal bones than of the spine. The compensation occurs in the change of the angle of the ribs and the change of the curve of the spine itself. The thoracic index, which is the antero-

<sup>2</sup> Dunn, H. L., *Anat. Rec.*, 1921, 21: 25-27.

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posterior diameter in relation to the lateral, is of importance in judging development and nutrition. At birth the anteroposterior diameter is 90 per cent of the lateral; by the end of the first year it is 80 per cent; by the eighth year 70 per cent, rising to 75 per cent early in maturity, which is the normal adult proportion.

At birth, the circumference of the chest at the nipple line, between full expiration and full inspiration, is a fraction of an inch less than that of the head. By the end of the third year these two relations are reversed. Generally speaking, however, for practical purposes throughout this whole period they are considered about equal. The relative measurements of the chest and head are given in greater detail in the following table from Holt:

AGE	SEX	CHEST		HEAD	
		Inches	Centimeters	Inches	Centimeters
Birth .....	{ Boys	13.4	34.2	13.9	35.2
	{ Girls	13.0	33.0	13.5	34.3
6 months .....	{ Boys	16.5	41.9	17.0	43.2
	{ Girls	16.1	40.8	16.6	42.3
12 months .....	{ Boys	18.0	45.7	18.0	45.7
	{ Girls	17.5	44.5	17.5	44.5
18 months .....	{ Boys	18.7	47.8	18.6	47.5
	{ Girls	18.2	46.2	18.0	45.7
2 years .....	{ Boys	19.3	49.1	19.2	48.7
	{ Girls	18.8	48.0	18.6	47.5
2½ years .....	{ Boys	19.8	50.4	19.5	49.5
	{ Girls	19.3	49.1	19.0	48.2
3 years .....	{ Boys	20.3	51.5	19.8	50.4
	{ Girls	19.8	50.4	19.4	49.3

During the first year of life the circumference of the chest increases 30 per cent, while during the second year the increase is only 10 per cent. From the second year to puberty there is a relatively slow development, at which time the anteroposterior diameter is 75 per cent of the transverse diameter. From puberty to the twentieth year there is a gradual development of structures and increase in size in all directions, but in the same relative proportions as at puberty. The size and shape of the chest are influenced to a marked degree by the state of nutrition, muscular exercise, and disease. Occasionally congenital deformities occur, some of which are hereditary.

**Muscles.**—The development of the muscles during fetal life is of little concern in the study of development and nutrition; in fact, very few observations have been made on the subject and perhaps fewer still

on postnatal development of these structures. The muscles of the body form about 25 per cent of the total weight at birth and increase to nearly 50 per cent at maturity. Thus it will be seen that the muscular increase in weight is about twice that of the body as a whole.

During early life the growth of the muscles of the trunk is greater than that of the extremities, while at maturity, the muscles of the extremities are more developed and form a greater proportion of total weight than those of the trunk. In this respect the lower extremity is developed to a greater extent than the upper. At birth, the muscles of the head and trunk furnish a little more than 40 per cent of the total muscular weight; in maturity they furnish only 25 to 30 per cent of the total muscle weight. During childhood the muscles of the lower extremity increase from 40 to 55 per cent of the total muscle weight. On the other hand, muscles of the upper extremity remain proportionately quite uniform, furnishing 18 to 20 per cent of the total muscle weight throughout life.

The actual anatomical development of muscles or the relation of their combined weight to the total body weight, is important since it is a definite index of the state of nutrition. This, however, is not demonstrable in the living subject. Muscle power, on the other hand, which is definitely influenced by the state of nutrition, amount of physical activity, and certain mental states, is of more relative importance in the state of development and nutrition. A lack of muscle function and a delay in the power of coördination is frequently a definite indication of mental retardation.

For the first few weeks or months of life, the action of the voluntary muscles is more or less purposeless. The arms and legs move, voluntarily, it is true, but with no evidence that such movements are consciously performed. The opening and closing of the eyelids may be regarded as voluntary but is rather instinctive; the act of closing occurs promptly when the eye is exposed to a bright light, in order to protect the eye. In some cases the infant may even turn the head away from a bright light for the same reason. The act of sucking is voluntary, but also is to be regarded as instinctive rather than intentional. The first real evidence of voluntary muscular action may occur as early as the end of the first week when the infant will often follow a light in the room by turning its head. This may be regarded as deliberate and purposeful.

By the fourth month, the infant makes deliberate efforts to grasp objects held before it, or plays with its own hands and feet (the first



real discovery). About the same time, often earlier, the head can be held erect independently, provided the trunk is supported. Much earlier than this, the infant raises its head from the pillow. By the seventh month, the normal infant can sit alone for a brief period and shortly thereafter for a longer time. At about the ninth month, the child begins to crawl and shortly after this, even at the same time, rises, assisted, on its feet and bears its weight for a moment. By the twelfth month it should stand quite well, held lightly by one or both hands. Average time of walking independently is about the fourteenth month. All of these developments are subject to individual variation within the limits of the normal and frequently show evidence of familial tendencies. The acquisition of these functions is most frequently delayed by malnutrition and long illness, but particularly by diseases intimately associated with nutritional (deficiency) disturbances, such as rickets. In delayed walking, lack of muscle power alone must not be considered; for lack of tendon tone and even unossified bones (rickets) play important parts. Less frequently, we find that retarded mental development is the cause of delay in such functions as the noticing of objects, holding up of head, sitting alone, and walking. After the first few years of life, muscular development increases as a result of the usual activities of child life, but is in very large measure dependent on the state of nutrition; in fact, the closest relation exists between nutrition and muscular development.

**Viscera.—Stomach.**—In recent years, during which more accurate methods of study have been devised, particularly through the use of the Roentgen ray, our ideas regarding both the shape and capacity of the stomach in young life have undergone radical changes. There is great variety in the shape of the stomach during the period of infancy, especially in the newly born. It has been found that in the newly born the stomach lies obliquely or even transversely in the upper abdomen, not vertically as formerly believed. The most common shape is that of the “inverted retort,” but other and quite common forms appear, variously described as “cow’s horn,” “fish hook,” or “sink drain.” However, it is the capacity of the stomach rather than its shape that concerns us most, since the increase of capacity is in direct proportion to the growth of the body as a whole.

There are two well-recognized methods of estimating the capacity of the stomach, known respectively as the anatomical and the physiological. The former, obviously, can be made only on the extracted, or static, stomach; the latter, of course, is made on the living subject and

shows the capacity of the dynamic stomach. Both are subject to objections. Physiological capacity is estimated only during the first year of life, while after this all data refer to anatomical capacity. In testing the physiological capacity, weighing before and after nursing is the only practical method. There are certain inaccuracies incident to this method which cannot well be avoided. The stomach may be over-distended; the esophagus may remain full, and a part of the ingested food may pass into the duodenum before nursing is completed. In spite of these objections, observations can be made on a large number of nursings in a single case and the average taken which is probably accurate enough for practical purposes. If such observations are made and checked by the x-ray, they are quite satisfactory.

During the first day of postnatal life, the capacity of the stomach is  $\frac{1}{4}$  of an ounce (7 c.c.); the increase is rapid, reaching  $1\frac{1}{2}$  ounces (45 c.c.) by the fourth day, while by the second week a capacity of 3 ounces (90 c.c.) is attained. The development of the stomach with consequent increased capacity from this time throughout the first year is quite constant, averaging from  $\frac{1}{2}$  to  $\frac{2}{3}$  of an ounce (20 to 25 c.c.) per month; so that by the sixth month it ranges from 10 to  $11\frac{1}{2}$  ounces (325 to 350 c.c.). In contradistinction to the average physiological gastric capacity, observations have been made to test the maximum gastric capacity. These observations are made on a series of unusually large nursings and average from 25 to 50 per cent more than the physiological capacity. After the first year, as already stated, studies on anatomical capacity furnish the only available data. These studies show that a capacity of 16 ounces (500 c.c.) is reached during the second year; a slight increase is shown during the third year, when the capacity is 18 ounces (550 c.c.), a similar increase occurring during the fourth year, when the capacity is 20 ounces (600 c.c.). From now on there is a gradual increase with quite marked variations until late childhood, when it varies from 25 to 30 ounces (750 to 950 c.c.).

*Intestines.*—At birth the length of the intestinal canal is subject to many individual variations, but a safe average is 158 inches (400 cm.). Of the total length, the small intestine furnishes approximately 132 inches (340 cm.), and the large intestine a little more than 24 inches (60 cm.). The small intestine increases in length 50 per cent by the end of the first year and is doubled at puberty. The large intestine a little more than doubles its birth length by maturity. The total combined length of the large and small intestines in the adult average 360 inches (914 cm.).

*Liver.*—At birth the liver weighs  $4\frac{1}{2}$  ounces (135 Gms.); it doubles its weight by the end of the first year, 9 ounces (270 Gms.), and trebles it by the end of the third year,  $14\frac{1}{2}$  ounces (405 Gms.). At the ninth year the birth weight has increased sixfold to  $1\frac{1}{2}$  pounds (810 Gms.), and by puberty tenfold to 3 pounds (1350 Gms.). The average weight of the liver at maturity is approximately twelve times its birth weight— $13\frac{3}{4}$  pounds (1620 Gms.). According to these figures it will be found that the liver furnishes at birth 4 per cent of the total body weight. During childhood generally, there is a decline in this proportion and it averages from 3 to 4 per cent of the total body weight, while in the adult the proportion is very close to 3 per cent. All statistics on weight and size of the liver are indefinite and quite unreliable. The weights are somewhat more reliable than the dimensions. The long diameter of the liver at birth is  $4\frac{1}{2}$  inches (11 cm.); the short diameter  $3\frac{1}{2}$  inches (8 cm.), and the thickness  $3\frac{3}{4}$  inches (8.5 cm.). In relation to the adult liver, these figures are approximately as follows: The length and breadth are about one-half of the adult measurement while the thickness is about two-thirds of that of the adult. The individual variation in the weight and dimensions of the liver is quite marked.

*Spleen.*—There is a very much greater variability in the size of the spleen than of the liver. The average weight at birth is  $\frac{1}{2}$  of an ounce (13 Gms.). Like a number of other viscera, its weight is doubled to 1 ounce (26 Gms.) during the first year and trebled at the end of the third year— $1\frac{1}{2}$  ounces (39 Gms.). From this time on the development is relatively slow but quite uniform and at maturity it is found to be about twelve times the weight at birth or  $5\frac{1}{2}$  ounces (156 Gms.). Accessory spleens are quite common and in some instances numerous; in fact, they are present in about one-third of all individuals. Their weight has not been considered in the previous figures.

*Kidneys.*—The combined weight of the kidneys at birth is  $\frac{3}{4}$  of an ounce (20 to 25 Gms.). This weight is doubled during the first six months and quadrupled by the end of the second year. At puberty the weight of the kidneys varies from ten to fourteen times their weight at birth. In the newly born the measurements of the kidneys are about as follows: Length  $1\frac{1}{2}$  inches (4 to 5 cm.); breadth 1 inch (2 to 2.5 cm.) and  $\frac{1}{2}$  of an inch (1.2 to 1.5 cm.) in thickness. These dimensions are increased by maturity to about two and one-half times. They are a little thicker in proportion during infancy than in adult life.

The stomach, liver, spleen, kidneys, and intestines show their

greatest rate of growth during the first year of postnatal life. From this time to the sixth year the growth is very slow, increasing slightly in rate from the sixth to the twelfth year, at which time there is a marked acceleration until we reach a rather definite period, between the fourteenth and sixteenth year, when the rate of growth is very rapid. There is an alternation of the growth of the viscera analogous to that of the bones; there is even an alternation in the growth of the cells making up the viscera.

*Bladder*.—The capacity of the bladder is variable and observations are quite inaccurate, largely because no reliable method has been devised for measuring it. According to the method employed the estimates vary from 1 to 1½ ounces (27 to 50 c.c.) in the newly born. This capacity increases steadily and is subject to many variations, particularly because of the ready distensibility of the viscus.

*Uterus*.—The development of the genital organs in the growing child is of no special interest in relation to nutrition. Physiologically, the development of the uterus is of interest. In the newborn, the average length is 1½ inches (35 mm.), subject to wide variations above and below this figure. The weight is usually about ⅛ of an ounce (3 to 4 Gms.). The cervix composes three-fourths of the total length and its walls are thicker than those of the body. This is known as the newborn type. In a short time after birth there is a diminution in both length and weight. This is known as the infantile type. The growth and development of the uterus is the slowest of all the organs. A slight growth is evident at about the fifth or sixth year but it does not return to the newborn type until the eleventh year. After this time a period of rapid growth ensues and by the establishment of puberty it shows all of the adult proportions.

*Mammæ*.—A fair analogy in development occurs in the case of the mammary glands to the uterus and bears a close relationship to development. Late in fetal life a number of tubules, showing some branching, begin to develop. This is the first indication of the mammary glands. There is no sharp differentiation between the ducts and the secretory portions and this is the condition found at birth. Within a few days after birth, however, the secretory portions of the lobules become physiologically active and secrete a milky substance, popularly known as "witch's milk." The physiology of its production is practically identical with that in the adult. There is engorgement of the breasts, accompanying the secretion. All these phenomena disappear by the end of the third week, but it is six months or more



before the ducts return to the natal state. During all this time, no distinction can be drawn anatomically or in size between the male and the female. From now on throughout childhood there is no change and still no differentiation between the sexes. Just before puberty, however, certain changes indicative of that period take place in the female. As this period approaches, there is an increase in size due largely to connective tissue and deposition of fat. At puberty the lactiferous ducts develop and the vascularity of the glands increases to a considerable extent.

*Thymus.*—The thymus is subject to a greater variation in size at birth than any other organ of the body even within presumably normal limits. Its average weight at birth is  $\frac{1}{2}$  of an ounce (13 Gms.), but by many observers is considered normal anywhere from  $\frac{2}{3}$  to 1 ounce (22 to 28 Gms.). It increases steadily until puberty, after which there is a slow decrease until some time in adult life. No data which are even approximately accurate are available on this phase of the subject. Generally speaking, the birth weight is doubled during the first five years and trebled during the second five years.

*Thyroid.*—Figures on the size of the thyroid gland, as is the case with a number of other organs of the body, are signally inaccurate. A great deal of the inaccuracy, in this instance, however, is due to the fact that most of the earlier investigations were made in the countries of Central Europe, in and around the Alps and, therefore, probably do not represent the normal thyroid. In older children especially, both weight and measurement are greater than in the average American child. Even in this country, there are a number of localities where a large thyroid is common, particularly during the prepuberal and puberal periods. At birth, the thyroid gland weighs between 1.5 and 2.5 grams, though variations slightly above and below these figures cannot be called abnormal without more exact knowledge on the subject. There is a gradual increase in the weight of the gland to 10 grams at the tenth year and  $\frac{1}{2}$  of an ounce (15 Gms.) at puberty. It is difficult to make a comparison between these figures and any available data concerning the gland in the adult, because of the great variation in its size and our insufficient knowledge at present of what is a normal gland. Noticeable enlargement of the gland, particularly in girls, occurs quite often during the puberal and prepuberal periods. There are certain sections of the country where such enlargement is more prevalent than in others, and yet its occurrence where no sectional influence can be considered as a rational cause is frequent enough for us to feel that

locality, kind of water, etc., are not the sole causes. How much of the enlargement at this period is to be considered pathologic and how much physiologic, due to increased activity of the endocrine system incident to puberal development, is not as yet determined.

*Heart.*—At birth the weight of the heart averages  $\frac{2}{3}$  of an ounce (20-25 Gms.), about equivalent to the combined weight of the kidneys. In its increase, the heart follows the approximate rule of the other viscera because the weight is doubled by the end of the second year and trebled by the end of the fourth year. At puberty the heart is about six times its birth weight. During adolescence, there is marked and rapid increase, so that at maturity it reaches a size varying from ten to thirteen times that of the newborn. It will be seen that at birth the heart is relatively heavier than it is in adult life. There is a decrease in the weight of the heart during the first year, but during the second year it rises to a weight closely approximating 0.50 per cent of the total body weight and this relation is maintained practically throughout life. Accurate data regarding the size of the heart in the growing child are scarce as in the case of most of the other organs, but according to the best available figures the following measurements may be taken as a fair average:

Age	Length, Centimeters	Breadth, Centimeters	Thickness, Centimeters
1 to 4 years .....	5.14	6.09	2.44
5 to 9 years .....	7.04	7.44	2.89
10 to 15 years .....	7.67	8.35	3.16

These dimensions are for boys; those for girls run from 5 per cent to 10 per cent smaller.

In the living subject even an approximate measurement of the heart in relation to the chest is difficult. Percussion varies with the individual examiner and with the posture of the child. The x-ray with a 7 foot plate, taken in an erect posture, gives us our most accurate observations at present, but even this is subject to error.

The growth of the heart is slower than that of the liver, spleen and kidneys during the first two years of life, after which it is much more rapid. For the first few months the child is relatively inactive and the growth of the heart correspondingly slow. Between the sixth and the ninth months, the child becomes much more active, with a relatively rapid increase in stature, and size of the heart. Then there is a period of relatively less activity during which the child is learning to stand

and walk, with a relative lessening of the growth of the heart. After the child learns to walk and run a period of great activity, extending from the third to the fifth year, ensues, during which both stature and heart weight increase rapidly. From this time on to puberty there is a steady increase in the size of the heart which is subject to slight periodic variations. But not until the beginning of the very active physiological changes incident to puberty does the heart make any very rapid increase in growth. This period is reached a little earlier in girls than in boys; consequently, the adolescent growth of the heart begins earlier in girls than in boys. "The weight of the heart, the size of the individual and physical activity are synchronous" (Bean).

*Lungs.*—The combined weight of the lungs at birth averages  $1\frac{2}{3}$  ounces (50 Gms.). This weight is doubled at six months and trebled at one year. From now on the rate of growth is quite steady and at maturity averages about  $2\frac{1}{8}$  pounds (1000 Gms.), which represents twenty times the weight at birth. The growth and expansion of the lungs play important parts in the shape and development of the chest and have in this way a secondary influence on posture. Exercise develops the musculature of the chest, but it also causes increased activity of the lungs producing greater expansion with a corresponding influence on the capacity of the thorax.

*Brain.*—The average weight of the brain of the newly born is 12 ounces (350 Gms.), but there is considerable variation in both directions within normal limits. This variation is relatively small, however, compared with that of other organs. The brain of newborn girls is slightly smaller than that of boys. The increase from birth to maturity varies from three and one half to four times.

There are definite cycles or periods in the development of the brain which are of importance in the handling of the growing child. During the first year, the growth of the brain is extremely rapid, its weight increasing about two and one half times in this period. This fact indicates that a great deal of rest and quiet, freedom from excitement and stimulation, are needed. From this time to the fifth year there is a decided slackening in the rate of growth but it is still quite rapid, acquiring a weight which is approximately three times the birth weight. This is a period of an acquisition of knowledge, largely through new functional experiences. After this the development in size, at least, of the brain is relatively slow and yet it reaches adult size by puberty. The brain of boys is actually, but not relatively, heavier than that of girls. The weight of the brain maintains a fair relation to the body

weight throughout the whole period of growth and this ratio is maintained throughout life in the average individual. Very nearly one-third of the total postnatal increase in the weight of the brain occurs during the first nine months and nearly two-thirds in the first two years.

*Cerebellum.*—The weight of the cerebellum at birth is  $\frac{2}{3}$  of an ounce (18 to 20 Gms.). From this time to maturity it attains a weight of  $4\frac{1}{2}$  ounces (126 to 140 Gms.), thus increasing about sevenfold.

Failure of the brain to grow, with resulting mental impairment, is due to a number of factors. Some of these are definite while others are theoretical. Heredity plays an important rôle: feeble-minded individuals, either with average-sized heads or heads markedly undersized, almost invariably produce offspring with like defects. This occurrence is in accord with the mendelian law and, therefore, there will be normal or abnormal offspring, according to the distribution of inherited factors. Mongolism shows a subnormal brain which is volumetric, while cretinism shows a deviation from the normal which is largely functional. Intra-uterine disease, particularly of the membranes, often produces abnormally small brains. During the period of postnatal development malnutrition often causes retarded mentality. How much of this retardation can be overcome by subsequent improvement in nutrition and how much is permanent as a result of prolonged malnourished states, it is impossible to state definitely with our present knowledge.

## PERIODS OF DEVELOPMENT OF ORGANS, TISSUES AND TRAITS <sup>3</sup>

**First Three Years.**—The first three years of life represent the period (postnatal) of rapid growth both actually and relatively. The muscular system is undergoing a radical and trying change, passing from a state of sluggishness amounting to almost complete passivity, to one of excessive activity. During this period, which we may term the period of acquisition of muscle power, the body movements are what are called large; they are the grosser or fundamental movements. In the early part of this period there is no sustained attention in making these movements; they are more or less purposeless. But as development progresses, the movements become intentional, coordinated, and more sustained. During most of this period, but particularly dur-

<sup>3</sup> For many valuable suggestions with regard to this section the author is indebted to Dr. Josephine E. Young.



ing the latter part, certain psychic phenomena appear and certain feelings or sensibilities develop. Some of these are considered instinctive, but may be evidences of imitation, or may be produced by environmental influences. The act of feeding is first automatic and almost a passive process but later becomes voluntary, and the liking or disliking of certain foods, or food biases, develop quickly, if not controlled. Also during these years, food habits, important factors in nutrition, are formed. Fear is usually an acquired trait, though due to stimulation of natural sensations. It is developed quite early during this period and requires most careful attention, especially in the direction of prevention or inhibition.

Anger shows itself early and is a manifestation of self-assertion and is to a certain degree desirable. Care should be exercised to teach control, but also not to break the spirit. Usually it is evidenced in crying, but more rarely it becomes resentful because of the development of selfishness during this same period. The imitative instinct is influential to a large extent in action, speech, and the acquiring of eccentricities, and in this respect these years are an important formative period. This instinct should be watched most carefully, since at the same time speech is being acquired and expression of thought, through speech, developing. The child is still dependent on the adult and cares little for other children and clings to parent or caretaker. Embarrassment becomes apparent in the latter part of this period and shows itself particularly in the presence of older people and even children, and, like the fear of other people, is caused by an awakening consciousness of self and the sense of dependence just alluded to.

Emotions not only manifest themselves but also indicate their trend quite perceptibly and must be safeguarded most carefully. Love looms large; it is bestowed lavishly and demanded in return. The latter tendency is the most frequent cause of a child being spoiled and this must be avoided. The first three years of life is the great period of child-spoiling. Imagination sometimes begins to show itself in the third year of life, though this varies considerably in different individuals. Memory is short; in a few hours or at the most in a few days, a child may even forget its mother. This is the great period of habit formation and it is exceedingly important to establish good habits, especially of bodily functions, at this time.

This is a period of rapid growth of the head and brain and one of the periods of activity of growth of teeth and jaws, carrying with it the shaping of the facial skull. The greatest spinal growth occurs

during this time, while marked development and growth of extremities occur in the early part of this period. During the first of these three years the lungs, liver, and intestines grow rapidly, due mainly to the assumption of the functions of respiration and alimentation as independent activities and also to the additional work put upon these functions by the remarkable increase in weight. The latter half of this period is especially remarkable because it is then that the greatest development of the heart occurs.

**Second Period (Four to Six Years).—**This is the period of the runabout. It is a time of continued growth and although coördination of muscle movements was begun in the previous period, true muscle control is slow in developing; hence, this age is one of awkwardness. Children begin to play with other children during this time but their attitude toward others is self-centered. They are learning what independence is, and consequently do not recognize the rights of other children at play. Imitation, which began to develop in the previous period, is now strongly developed and forms the chief motive of habit formation. Curiosity becomes a striking characteristic and imagination almost the controlling impulse in play and thought. The child being self-centered and selfish wishes to gain possession of every article in sight. To this is added a pride of possession which makes this period important in teaching the doctrine of "mine and thine" and respect for honesty of purpose and possession. The formation of habits, which is the repetition of things done before, continues from its beginning in the previous period, but with greater intensity and calls for the strictest control. During this period the child is very active in asking questions, a manifestation of curiosity; the noticing of things and phenomena and the desire to know about them. The answers given to questions and the explanation of phenomena should be carefully studied and the truth should always be dominant.

The head continues to grow, not so rapidly in the cranial portion, but largely on account of the development of the paranasal sinuses there is a more rapid relative growth of the facial skull. The sixth year marks the second period of skull development. During this period, that is, by the end of the fourth year, the natal weight of the heart is trebled, while by the end of the fifth year the brain is three times the birth weight. By the middle of this period, the birth weight of the thymus is doubled.

The rapid growth begun in the first period continues practically through the second, and the latter part of this period represents the

second period of rapid growth of the torso; the heart, liver, spleen, head and pelvis growing rapidly. The spine shows a well-marked anterior concavity in the dorsal region and a corresponding lumbar convexity towards the end of this second period. During the early part of the second period there is a marked acceleration of the growth of the extremities. At the end of this period, the trunk is 30 per cent of the total stature; the lower extremities 45 per cent and the upper 40 per cent. The first molar tooth shows at the end of this period. The capacity of the stomach reaches 600 c.c. and a very slight growth of the uterus takes place about this time. At the end of this period and overlapping well into the beginning of the next, the self-centeredness of the child demands for itself a position in the center of the stage.

**Third Period (Seven to Ten Years).**—The development of the muscular system continues, although motor activity is somewhat less. The beginning of coördination, which we saw take place in the second period, advances rapidly. Dexterity is acquired rapidly and the combination of coördination with dexterity results in a considerable amount of skill. Through this combination of forces, actual muscle power, that is, strength, develops. Self-consciousness developed during a previous period, is transformed into an intense individualism, with the result that the child acquires an overbearing spirit towards others and patronizes those slightly younger or smaller. This individualism is also accompanied by a definite amount of determination, the combination resulting in interesting competition in games and sports. Intense individualism is gradually modified and eventually merges into the *individual group* which develops the gang spirit. It will be seen, therefore, that this period is one of critical transition and adjustment and is often exceedingly difficult to manage. Gentle, firm, and judicious guidance is called for constantly at this time. During the latter part of this third period an abrupt change occurs; childhood is lost and youth begins. Up to this time the child has acted almost entirely from imitation and through force of habit and largely without thinking. When this change occurs the mental processes characteristic of the adult develop. Conduct is guided more by reason and a logical sequence of events begins to control conduct.

This is the period of sex awakening. Observations which have been made by those interested in this phase of child life, aided by statements of numbers of adults relative to their remembered experiences, have shown quite clearly that such an awakening occurs usually about the seventh year. Sex consciousness and even sexual stimulation appears

earlier in girls than in boys. These facts are strongly at variance with popular opinion and it is regrettable that erroneous ideas are entertained along this line by a large majority of the medical profession. There are but two primal instincts—hunger, which calls for relatively slight guidance, and the sex instinct which should be guided carefully and intelligently from its very beginning.

The head grows, the bones increase in thickness, and the sutures of the skull become ossified. The paranasal sinuses are expanding rapidly, with a consequent rapid development of the facial skull, this assuming for the first time the resemblance to the adult type. There is a calcification of the third molar teeth; the permanent incisors appear early in this period and the bicuspid late in the same period. About the middle of this period a rapid growth of the extremities occurs. The liver is six times heavier than it was at birth. The anteroposterior diameter of the chest is 70 per cent of the lateral diameter; closely approximating the adult type.

**Fourth Period (Ten to Sixteen Years).—**During the early part of this period physical energy reaches its peak and declines slowly during the balance of life. The coördination of muscle control and the acquiring of a deliberateness in thought continue to develop rapidly, and skill continues to increase remarkably. The period of youth is well developed, showing all of the consciousness of approaching maturity. Children learn rapidly, grasping with avidity knowledge concerning any new object or line of thought. Individualism temporarily merged into the group in a previous period reasserts itself and, though the group spirit may continue, the individual makes strenuous efforts in physical or mental competitions to outstrip contemporaries. During the later part of this period there is a rebirth of the emotions. Self-consciousness is again apparent, shown through shyness and awkwardness in conduct which is mental and not physical, as before. It is the time of the forming of intimacies between those of the same sex or of the opposite sex. It is a period of the birth of romance and of hero-worship. Mysteriousness is attractive and the formation of clubs which meet behind closed doors is a common pursuit. Secretiveness is a prominent characteristic and is more apparent with girls than with boys. Especially with the former class, there is an apprehension of something expected but not understood. This period, in fact, presents the most abrupt change of mental attitude in the whole history of individual existence. It is a period during which the children of both sexes develop mental biases hitherto unexpected. Hysterical manifesta-



tions of minor character display themselves and are especially liable to occur in children unfortunate enough to have inherited a neurotic taint, or those who have been reared in homes of domestic unhappiness, or in those homes where luxury and leisure have supplanted profitable vocations and ennobling pursuits.

This is the last period of rapid growth of the torso; broadening of the body takes place and a marked enlargement of the pelvis reaching almost adult proportions. The brain, kidneys and lungs grow rapidly; the liver and spleen reach their mature weight, twelve times the birth weight. The uterus returns to the newborn size and in the last year of this period reaches full adult proportions. This is the last period of skull development, largely through the rapidly expanding facial sinuses. The extremities are growing rapidly; the lower extremity reaching 50 per cent of the total stature and the upper extremities 45 per cent. The canine teeth are fully developed between the twelfth and fifteenth years, and the second molars about the same time. There is less curving of the dorsal and cervical spine but a marked lumbar curving appears. At puberty the heart has reached a weight which is six times the birth weight. In the female during this period, the breasts begin to enlarge and the figure assumes the early adult type.

During the early part of this period, both in boys and in girls, but particularly the latter, there is a tendency with some to become exceedingly obese just at the time that the average child is more slender. The figure of such children reminds one of the middle-aged individual and bears a strong resemblance to figures ascribed by endocrinologists to derangements of the endocrine system. It is entirely likely that the changes incident to sex development produce a hormone which influences one or another of the endocrine glands. In some instances these individuals continue to be obese with unusual local deposits of fat. In other instances, however, they become slender on the full development of puberty and carry normal weights through the balance of their lives.

## CHAPTER VI

### GROWTH

As a result of the development of the various organs of the body as just described, we have the phenomenon of growth, which term is used to designate increase in stature. Weight in relation to height must be considered to some extent under growth, but will be mainly dealt with in our discussions of the normal child.

The underlying causative factors responsible for growth have been subject to much speculation and many theories have been advanced in an attempt to explain the phenomenon. There are probably two or more definite causes, among them being a chemical process and a physiological one. The chemical process occurs in the course of and incident to metabolism. It is probable that some chemical change, while in progress, has the power of accelerating further chemical changes. This is known as the theory of self-producing catalyzers. It is known that growth occurs in cycles which begin slowly and become accelerated in velocity as they progress, up to the point of greatest activity, which is the peak of the cycle; then follows a gradual retardation, which, however, is more rapid than the period of acceleration and immediately precedes the institution of another growth cycle. The catalytic theory explains this through an accumulation of the stimulating process to a point where there is an exhaustion of activity.

The physiological process acts through a multiplication of cells; by division, by enlargement of individual cells, by intussusception (reception or ingestion of food); and by the deposition of intercellular tissue. There is an alternation in cell division and enlargement parallel with and analogous to the enlargement and growth of the tissues and organs of which the cells are a part.

During antenatal life, the individual is either a part of its mother or a parasite, according to the point of view from which we regard it. Its existence is dependent and essentially parasitic. At the moment of birth, however, an abrupt change occurs, and the fetus becomes, physiologically, an independent organism and continues from then until maturity to grow in stature and weight according to a definite curve which is subject to individual and periodic variations.

The average weight of a child at birth is 7.35 pounds (3.33 Kgs.) and the average length is 20.5 inches (52.2 cm.). These figures represent a mean between the averages of male and female infants; males being slightly more than this and females slightly less. There is marked disproportion between the length of the trunk and the lower extremities, which is best shown by the fact that the half height at birth is at or a little above the umbilicus; while in the adult it is just about the upper part of the symphysis.

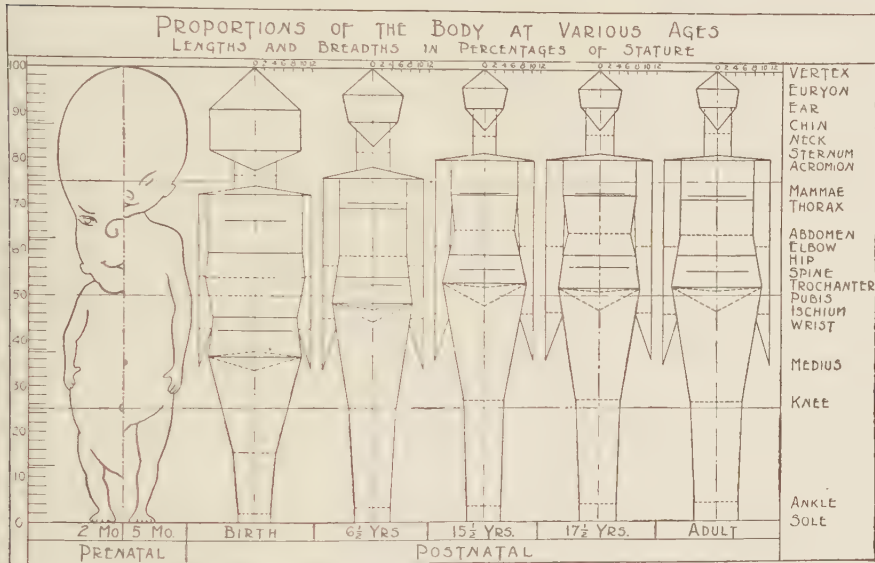


FIG. 6.—DIAGRAMS ILLUSTRATING THE CHANGING PROPORTIONS OF THE BODY AT VARIOUS AGES.

(From Bean, modified from Stratz and Godin. From *Anatomical Record*, Vol. 28, No. 1.)

During the next six months the average child grows approximately six inches (15 cm.) and a little more than doubles its birth weight, measuring at this age 26.2 inches (66.7 cm.) and weighing 16 pounds (7.15 Kg.). By the end of the first year the child measures 29 inches (75 cm.) and the birth weight is approximately trebled, being 21 pounds (9.5 Kg.). From this time on to the end of the third year, there is an increase in height of 8 inches (20.2 cm.) and a gain in weight of 11 pounds (5 Kg.) so that at the end of the third year the child is 36.8 inches (93.4 cm.) tall and weighs 31.5 pounds (14.31 Kg.). During the second six months the gain in weight is practically the same as in the first six months in actual amount, but during the first six months the

increase is 100 per cent, while during the second six months it is only 50 per cent, showing a marked decrease in the rate, which decrease continues through the second and third years, when the rate of increase is 25 per cent for the second year and 20 per cent for the third year. The percentage of increase in height diminishes constantly as age advances. During the first six months there is an increase of 21 per cent and during the second six months 14 per cent. The percentage of increase diminishes continually until maturity.

It is usually stated that artificially fed infants average less in weight than those who are breast fed. This is true with the ordinary dilutions of cow's milk, but infants fed on undiluted acidified milk, with the addition of sugar, in the experience of the author, weigh as much as those fed naturally.

As we have seen, the most accurate single guide to a state of nutrition is the relation of weight to height. The growth in both weight and height during the first three years is so uniform in relation to age that for all practical purposes during this period weight to age is the basis of computation except in very small (premature) infants. After the third year only weight to height is considered. A growth in height and an increase in weight during the first three years of life are best shown in the following table, taken from Holt:

AGE	SEX	WEIGHT		HEIGHT	
		Pounds	Kilograms	Inches	Centimeters
Birth .....	{ Boys	.55	3.43	20.6	52.5
	{ Girls	7.16	3.26	20.5	52.0
6 months .....	{ Boys	16.0	7.26	26.5	67.4
	{ Girls	15.5	7.03	26.0	66.1
12 months .....	{ Boys	21.0	9.54	29.5	75.0
	{ Girls	20.5	9.31	29.0	73.7
2 years .....	{ Boys	24.5	11.13	31.5	80.0
	{ Girls	23.7	10.77	31.0	78.8
2½ years .....	{ Boys	29.7	13.50	35.5	90.2
	{ Girls	28.7	13.04	35.0	89.0
3 years .....	{ Boys	32.0	14.54	37.0	94.0
	{ Girls	21.0	14.09	36.5	92.8

A large number of children have been studied both abroad and in America with reference to the ratio between age and height. These studies have been made by a number of observers and on children of various nationalities and from different social environments. As



might be supposed, the plotted curves representing the average heights differ according to the group of children studied. The observations made in foreign countries show a decidedly smaller stature for the corresponding age than that of children observed in America. The height curve for age on American children varies also according to whether the observations were made on private-school children or those of the public schools, and even among public-school children in different parts of the country. All heights so considered are obviously standing heights.

The following table is based upon the work of the National Child Health Council and is recommended by them as a safe, practical guide :

Age	Boys		GIRLS	
	Height, Inches	Height, Centimeters	Height, Inches	Height, Centimeters
48 months .....	39	99	39	99
60 months .....	41.5	105.3	41.5	105.3
72 months .....	44	111.5	44	111.5
5½ to 6½ years ..	46	116.7	45	114.2
6½ to 7½ years ..	48	121.8	47	119.3
7½ to 8½ years ..	50	126.9	50	126.9
8½ to 9½ years ..	52	132	52	132
9½ to 10½ years ..	54	137.2	54	137.2
10½ to 11½ years ..	56	142.1	56	142.1
11½ to 12½ years ..	58	147.2	58	147.2
12½ to 13½ years ..	60	152.3	60	152.3
13½ to 14½ years ..	63	159.8	62	157.4
14½ to 15½ years ..	65	165	63	159.8
15½ to 16½ years ..	67	170.1	64	162.5

All figures showing the relation between height and age are subject to great individual variation. In some observations this variation amounts to as much as 6 inches at five years of age and 10 inches at sixteen. Whether the figures at the extremes of these variations can be considered as representing normal children is open to serious question and, therefore, the increment of growth at various ages is in many respects a safer guide than a comparison with the average. We find that this increment varies between 2 and 3 inches for the period from the fifth to the sixteenth years and may be stated as 2½ inches as a fair average. The average annual increase in weight and height at different ages is well shown in the following tables taken from Holt, *American Journal of Diseases of Children*, Volume XVI, page 374 :

## GROWTH

## BOYS

AGE, YEARS	WEIGHT		HEIGHT	
	Range, Pounds	Average, Pounds	Range, Inches	Average, Inches
6-7 .....	3 - 5	4	1.5-2.5	2
7-8 .....	3.5- 5	4.75	1.5-2.5	2
8-9 .....	4 - 6	5.25	1.5-2.5	2
9-10 .....	4 - 7	6	1.5-2.5	2
10-11 .....	4 - 6.5	5	1.4-2.0	1.7
11-12 .....	4.5- 7.5	6.5	1.3-2.0	1.8
12-13 .....	6 - 9	8	1.5-2.5	2
13-14 .....	8 -12	10	2 -3	2.5
14-15 .....	10 -15	12.5	2 -3.5	2.7
15-16 .....	9 -15	13.75	2 -3.5	2.7
16-17 .....	4 -12	6.5	0.5-2	1.2
17-18 .....	4 - 8	5	0 -1	0.5

## GIRLS

AGE, YEARS	WEIGHT		HEIGHT	
	Range, Pounds	Average, Pounds	Range, Inches	Average, Inches
6-7 .....	3 - 5	4	1.5-2.5	2
7-8 .....	2 - 5	4.5	1.5-2.5	2
8-9 .....	3.5- 6	5	1.3-2.4	1.75
9-10 .....	4 - 6	5.25	1.5-2.5	2.25
10-11 .....	4.5- 7	6.5	1.5-2.5	2
11-12 .....	7 -11	9.5	2 -3	2.5
12-13 .....	8 -12	10.5	2 -3	2
13-14 .....	7 -11	9.5	1.3-2.4	2
14-15 .....	5 -10	7.5	1 -2	1.25
15-16 .....	3 - 8	6	0 -1	0.75
16-17 .....	2 - 6	3.5	0 -0.5	0.50
17-18 .....	0 - 2	0.5	0 -0.2	0.2

The height and age curves of various observers are shown on the chart on the next page, taken from Benedict and Talbot, *Metabolism and Growth*, Carnegie Institution.

Comparisons of the rate of growth at various periods from the moment of fructification of the ovum throughout life show that the *peak* of growth is reached during early intra-uterine life; in fact, the maximum rate is during the first month. After the peak is reached, the *rate* of growth declines steadily, and as far as growth and development are concerned senescence commences at this time. With the exception of the remarkable phenomenon just mentioned, the period of most rapid growth in weight is during the latter part of pregnancy,

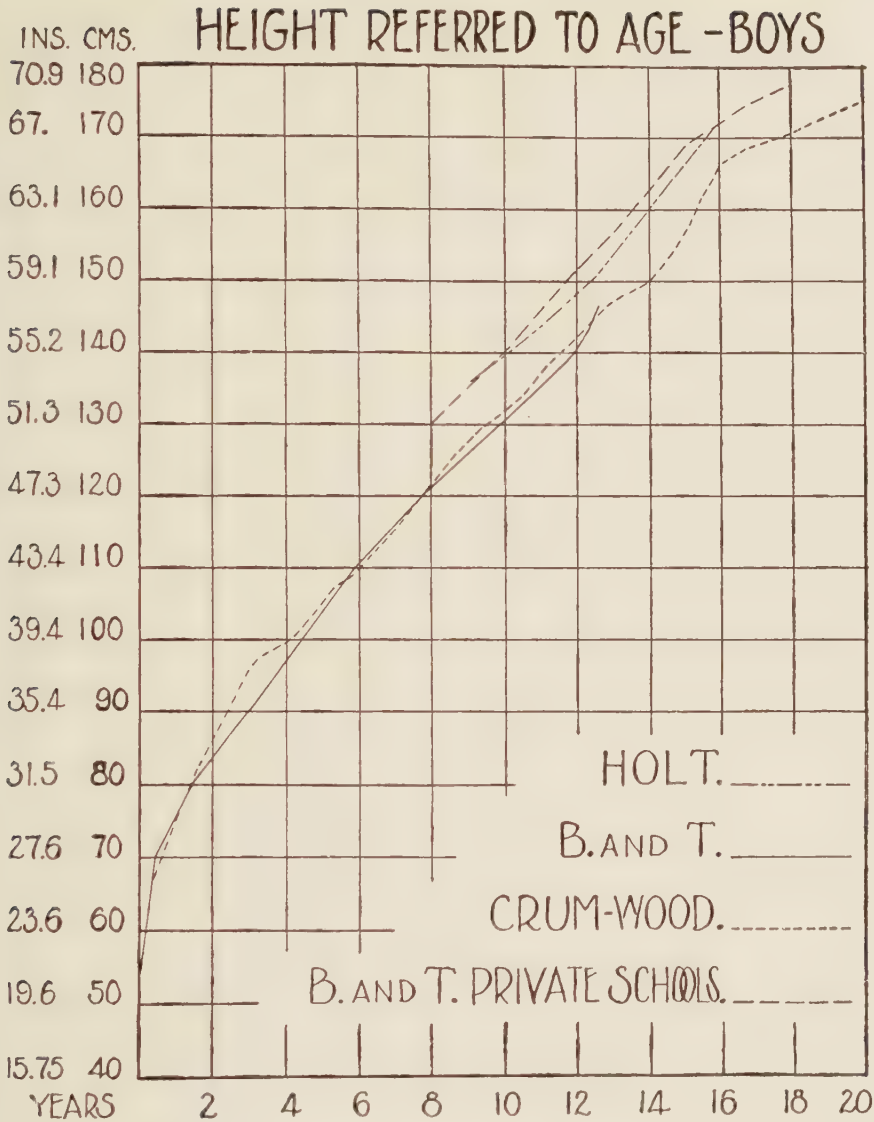


FIG. 7.—DIAGRAM SHOWING THE RELATION BETWEEN HEIGHT AND AGE FOR BOYS.  
(Adapted from Benedict and Talbot, *Metabolism and Growth from Birth to Puberty*,  
Carnegie Institution, Washington, D. C.)

and the rate continues to be rapid during most of the first year of post-natal life. During the closing months of the first year, however, there is a marked slackening of the rate of growth, which continues slow throughout the second year. Then comes a period of more rapid

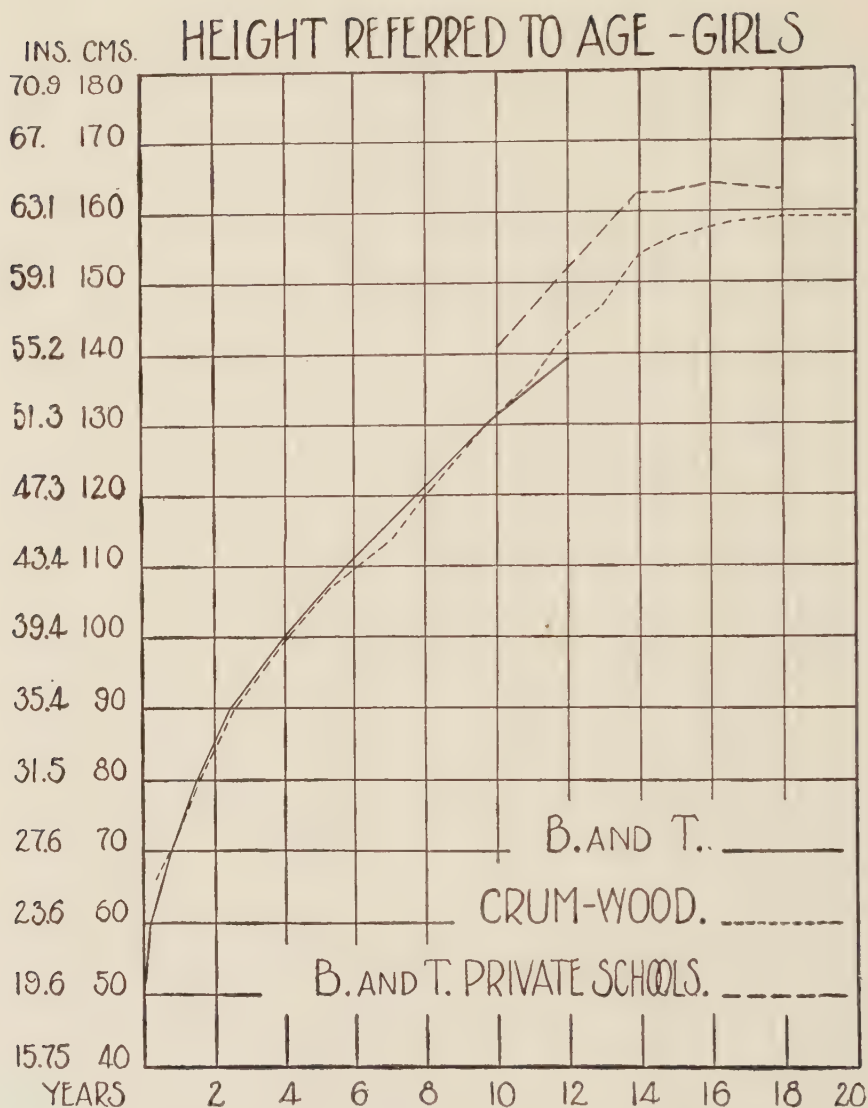


FIG. 8.—DIAGRAM SHOWING RELATION BETWEEN HEIGHT AND AGE FOR GIRLS.  
(Adapted from Benedict and Talbot, *Metabolism and Growth from Birth to Puberty*,  
Carnegie Institution, Washington, D. C.)

growth, continuing through the third, fourth, and fifth years, when another lull occurs which immediately precedes the onset of the very rapid and vigorous growth of adolescence. The rate of growth is well shown by the fact that during the first five years the body increases in length by 100 per cent; while during the next five years the increase is only



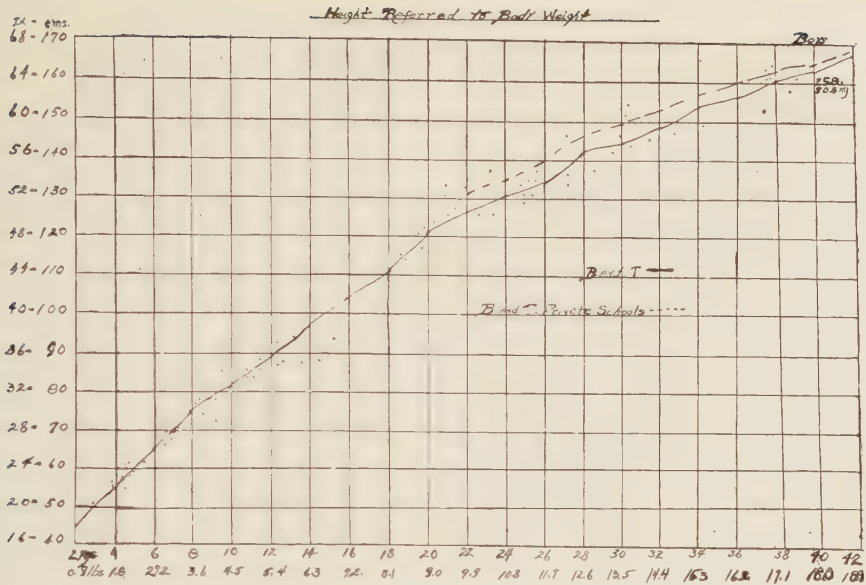


FIG. 9.—DIAGRAM SHOWING RELATION BETWEEN HEIGHT AND BODY WEIGHT IN BOYS.

Note variation of individual weights on each side of average curve. (From Benedict and Talbot, *Metabolism and Growth from Birth to Puberty*, Carnegie Institution, Washington, D. C.)

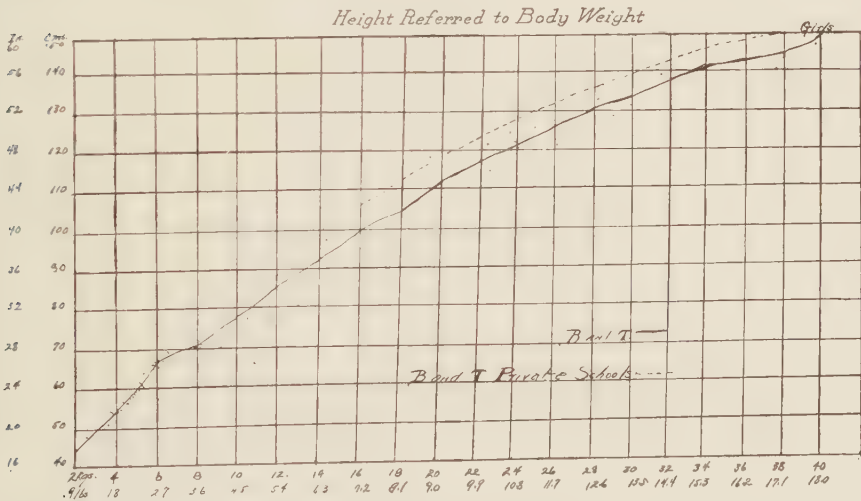


FIG. 10.—DIAGRAM SHOWING RELATION BETWEEN HEIGHT AND BODY WEIGHT IN GIRLS.

Note variation of individual weights on each side of average curve. (From Benedict and Talbot, *Metabolism and Growth from Birth to Puberty*, Carnegie Institution, Washington, D. C.)

50 per cent. After that the rate is not so constant and is subject to the influence of many factors. Growth is manifested by a definite average curve, but is subject to many individual variations.

The chief growth of stature, for the whole period, is just before puberty, that is, in late childhood; while the chief increase in weight is just after puberty, or at the beginning of maturity. At puberty there appears to be a rebirth of all growth, with a reproduction of rate and cycles. Periods of rapid growth of stature represent the periods of growth of bone, already described, and these alternate with the periods of the growth of teeth. Bone growth is particularly active at puberty.

There is relatively rapid growth of the extremities, particularly the lower ones, before puberty and of the trunk after puberty, so that before puberty, increase in weight is relatively greater in the osseous system, and after puberty it is relatively greater in the muscular system. The growth of the extremities in proportion to the trunk varies as age advances.

At birth the trunk is about 40 per cent of the total stature, and the lower extremities about 35 per cent. The arm length at this time is 38 per cent of the stature, being longer than the lower extremity, but both are shorter than the trunk. Between the sixth and seventh years the trunk length is 30 per cent of the stature, the lower extremities 45 and the upper extremities a little over 40 per cent. Thus the extremities are longer than the trunk and the lower extremities are longer than the upper. The proportion of the trunk remains practically the same from now on—*i.e.*, 30 per cent. At or near puberty the lower extremity is a little more than 50 per cent of the stature and the upper extremity 45 per cent. These figures are very nearly the proportions found in the adult. A striking comparison in the changes in proportion is found in the fact that at the second month of intra-uterine life the head is as long as the rest of the total stature, while in the adult the half height is at or near the upper border of the pubes. These facts are exceedingly well shown in the diagram (Fig. 6).

In studying a large number of children, especially those taken from public schools, it will be seen that although an average height for age may be stated, there is a marked variation from this average in the individuals observed. In some individuals there is a variation of as much as 7 inches on either side of the average. There are a great many factors which influence the height of the individual and must be considered when we study a single child. Mention has already been made of the fact that observations made in different countries show a widely

differing average curve for height in relation to age, and that each nationality has its characteristic height-age curve. In any study of this kind, therefore, the racial influence must be reckoned with. Familial traits are well recognized. These traits are considered in the light of biological cell determiners for tallness or shortness, certain families showing a general tendency to be tall while others show a similar tendency to be short. Where two families have been united, one characteristically tall and the other characteristically short, some of the progeny may be short, some tall, and others may be intermediate between short and tall. All of these heights are accounted for by the mendelian law. But what the influence is for tallness and shortness in these families originally is not accounted for. In a country such as the United States, where there is often a mingling of races with consequent intermarriage, the racial factor must be considered in studying public-school children, particularly in large centers of population.

Even within the confines of a single large nation such as that of the United States, we find more or less definite localities in which the people are tall or short, relatively speaking. For example, the mountainous sections of one of the states is noted for its men and women who are noticeably taller than those of other parts of the country. To explain this phenomenon would call for a type of speculation totally unwarranted with our present knowledge. Social status of the family from which a child comes must always be considered when studying its rate of growth. This social status, however, largely resolves itself into the economic status. In the lower strata of the social scale, we are apt to find that the food has been inadequate in both quantity and quality; that the children are required to do arduous work at an early age and that medical attention is not evoked sufficiently early to prevent either serious illness or a state of malnutrition. As a result of this, the children are apt to be not only undernourished but to some extent stunted in their growth. In the higher strata of this economic scale, however, the reverse of these conditions usually exists and children of this class are generally taller for their age, though not uniformly heavier in relation to their height.

We have seen that the phenomenon of growth is dependent on certain chemical and physiological processes, and this is true; but both of these forces are dependent on the food supply and this presupposes a balanced ration in which there is an adequate amount of protein to take care of the development of the human organism, and a sufficient number of calories for the needs of the body. A balanced ration contains a

sufficient amount of vitamins which are the growth factors in the food, and these vitamins to a large extent determine growth in stature. Growth in weight is largely dependent on the total number of calories. But growth in height cannot take place through the administration of vitamins alone but only in the presence of a sufficient total food supply. The glands composing the endocrine system probably play an important part in the regulation of growth. The pituitary gland, or, at least, one portion of it, is at present supposed to perform this function. Another of the endocrine glands, the thyroid, at least influences the metabolic rate to a considerable extent. In what manner the hormone from each of these glands performs the function credited to it or by what they are stimulated, is not as yet known. It is entirely likely that some combination of food elements is responsible for this stimulation.

Benedict and Talbot state that a suppression of growth by deficiency of food is followed by a subsequent catching up and that growth in stature is not interfered with but only the state of nutrition. Whether this is a final statement of fact or not is probably still debatable, but their secondary conclusion is in a broad sense probably correct; that if a child is not up to normal height for age we may presume that food accessory substances, or vitamins, have been deficient. We may presume a deficiency in calories where weight alone is below normal; in other words, where it has not been sufficient to take care of the heat output. Such statements as these are clearly dependent on whether we are studying a child from a certain class and comparing it with the average of that class.

It is well known that malnutrition during the first year of life bears a very important relation to malnutrition during the subsequent years of childhood. It is not known at this time, for lack of competent observation, to what extent, if any, stature is influenced by this early malnutrition. We have already noted the influence of sunshine on metabolism, and also that of some of the earthy salts. This same influence is apparent in the seasonal alternation in the periods of growth. Observations along this line have been rather limited and such as have been made are during the later years of childhood, specifically the period from the seventh to the fifteenth year. Observations on the seasonal rate of growth have resulted in conclusions differing in many important respects, but show quite conclusively that a marked seasonal alternation occurs. The two statements which follow represent our knowledge on this subject as well as any. Tanner, quoted by Chapin, states that the period of most rapid increase in height is from April to



August, and the period of slowest increase is from August to December; that the greatest increase in weight is from August to December and the smallest increase from April to August. Holt, on the other hand, states that from November to May is the period of least growth and from May to November the greatest; and that from May to November it was also found that an average of  $1\frac{1}{4}$  pounds more was gained by a child than during the other period. The rate of growth in stature during the summer months was 0.36 inches greater than during the winter season.

## CHAPTER VII

### THE NORMAL CHILD

When an attempt is made to state in dogmatic terms what constitutes a normal child, one is confronted with a serious and difficult problem. At present there is a lack of data derived from adequate observations by which we may arrive at any conclusion more than approximately accurate, surely none approaching finality. The point of view, or bias, of one observer differs materially from that of another, and preconceived notions, or ideas, of what the normal should be influence deductions to no small degree. Almost all published observations have been made on large numbers of individual children and the results averaged and this average has been assumed to be normal. Again we are confronted with the question considered in the chapters on Development and Growth as to whether we should regard average children from various social strata, possessing in many instances racial admixtures, as normal children, or whether we should regard only an "ideal" as normal. In this connection "ideal" refers to children from a high economic social level and reared under relatively "ideal" conditions. It is not at all unlikely, however, that as our knowledge advances and more minute observations are made, it will be found that what are considered "ideal" conditions at present do not produce children who are in reality "ideal." As yet we do not know what "ideal" living conditions are. It is exceedingly doubtful whether the modern steam-heated, practically air-tight, and consequently ill-ventilated dwelling (in which type of house those of the higher economic social strata reside), is a suitable environment for the growing human organism. It is quite conceivable that an open-air, tribal life, accompanied by an adequate food supply might well conduce to the development of a superior type of individual.

A standard of normality, therefore, for the present must be established from the standpoint of relativity, due consideration being given to an ideal and comparisons made with an average. Ultimately the decision rests with the individual examiner, who must be guided by certain criteria, based on the observations and judgment of those who have had large and varied experience in applying fundamental principles.

Anthropologists determine normality largely from a morphological standpoint based on anthropometric observations; physiologists have as their guide the rate of basal metabolism; psychiatrists regard the child almost entirely in the light of mental capacity; the physician and public health agencies largely in terms of acute illness and acute communicable disease. The pediatrician regards the child as a whole, and in order to do this must view it from each of the points of view mentioned. Since observation along all these lines would necessitate examination by a group of specialists, it is obviously impracticable to determine to what extent the child is normal in every sense of the word. It is necessary, therefore, that the family physician, school examiner, or public health nurse, be supplied with an outline of the more important attributes of the normal child, to serve as criteria in determining whether a given child is approximately normal or how far such a child deviates from the normal.

The present work deals with normal development and growth and the normal state of nutrition and with the abnormal only in relation to nutrition. Pathological conditions due to infections and other disease entities will be considered only in so far as they bear directly on the state of nutrition. In any observation made to determine the state of a child's nutrition it is just as necessary to make a complete physical examination as if we were endeavoring to make a diagnosis in an acute illness; therefore, all discussions relating to normal or abnormal states of nutrition presuppose a complete physical examination. In this series, infant feeding is discussed in a volume devoted entirely to that subject, and yet the normal infant from a nutritional standpoint must be considered briefly in this volume, as a foundation for the discussion which follows.



FIG. II.—NORMAL INFANT FIVE MONTHS OLD.

Birth weight was seven pounds eight ounces; present weight sixteen pounds eight ounces. Child sits alone, supporting itself, but can sit without support for a moment or two. Has bright appreciative countenance.

The weight and length of the newly born infant depend to a large extent on how long the infant has remained in utero—the duration of gestation. Other conditions influence the size of the newly born, such as disease in the mother and certain intra-uterine diseases of the fetus itself, the latter presupposing the former. During the first few days of life, there is an initial loss in weight, due to the removal of the vernix caseosa at the first cleansing; to loss of water from the kidneys and



FIG. 12.—INFANT SIX MONTHS OLD.

Birth weight was seven pounds four ounces; present weight fourteen pounds ten ounces. Sitting unsupported.

lungs; to the bowel discharges, composed entirely of meconium, containing itself a certain amount of water. During this same period, as a rule the child secures no food from the mother and a relatively small amount of water is taken; hence there is no compensation for the loss referred to. Under normal conditions this initial loss reaches its lowest point at the end of the third or fourth day. There is a relatively greater initial loss in premature infants and those congenitally weak (physiological prematurity). Available data show a rather striking variation in the time at which birth weight is regained. Taking all statements into consideration we find that the average is somewhere be-

tween the tenth and fourteenth day, and differs with individual children, and according to whether the mother is a primipara or a multipara and, of course, depends in a large measure on the initial supply of mother's milk. It has been shown by a number of observers that the initial loss of weight can be prevented in some measure by beginning the administration of both food and water within a few hours after birth. Observations along this line have been made during the last two years on the newly borns in the University of Virginia Hospital, and it has been shown that not only can the initial loss be reduced materially, but that by complementing the supply of mother's milk



during the early days, infants have regained their birth weight in an average of eight days. The effect of the amount of initial loss and that of the time of return to the normal birth weight on the subsequent development of the child have not yet been determined. It is the impression of those who have observed these experiments that subsequent gain in weight during the early months of infancy has been more steady than in those infants with greater initial loss of weight and a prolonged return to birth weight. Whether this procedure merits serious consid-



FIG. 13.—SAME CHILD AS IN FIG. 12.  
Shows muscle power and mental alertness.

eration in the child of average weight or above the average weight at birth, is perhaps doubtful; it undoubtedly improves the state of nutrition in premature infants and those full-term infants who are much below the average birth weight.

We have seen that the average length of a normal infant at birth is 20.5 inches (52.2 cm.), and the average weight for both boys and girls is 7.35 pounds (3.34 Kg.). At six months the height averages 26.3 inches (66.8 cm.), and the weight is nearly 16 pounds (approximately 7 Kg.). At one year the height is approximately 29 inches (74 cm.), and the weight about 21 pounds (or 9.5 Kg.). So large a percentage of infants follow this rate of increase in both height and weight so closely that a curve based on these averages, for the first twelve months may be considered a fairly safe guide. It will be seen from this that the growth in stature for the first six months is approxi-

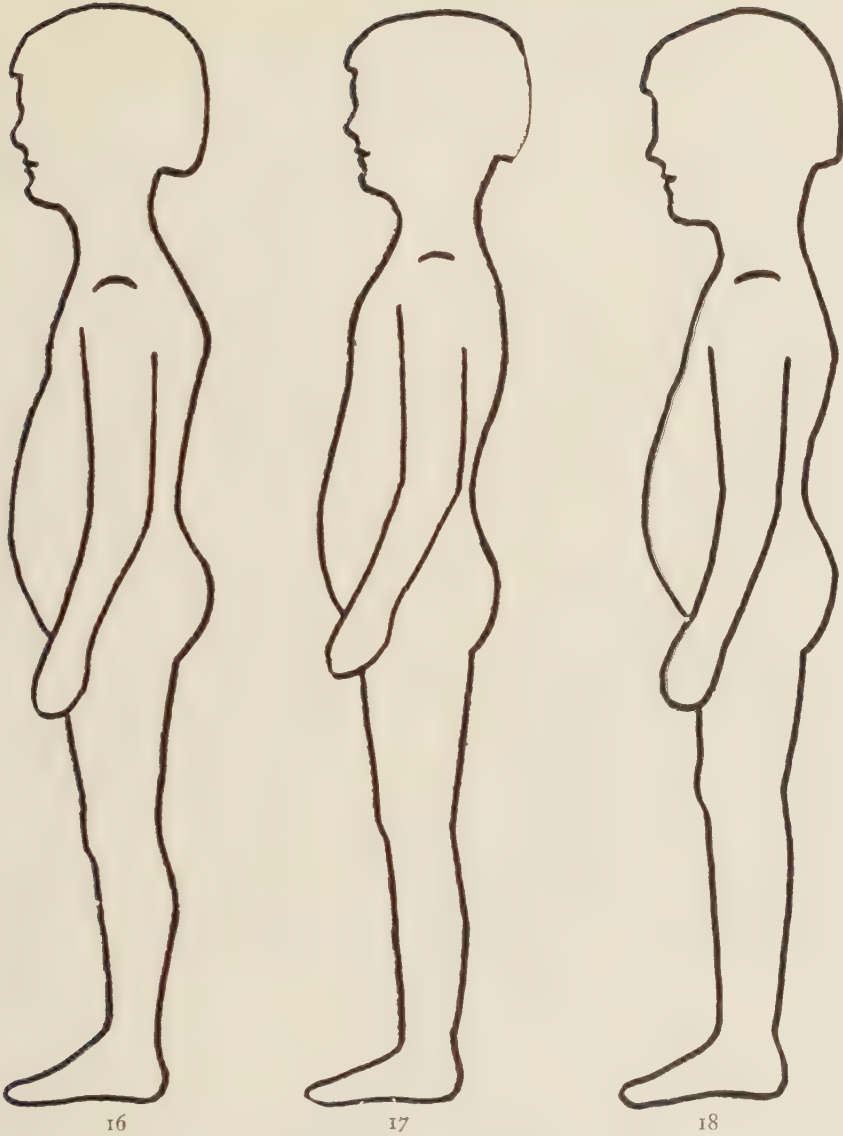
mately 6 inches and for the second six months 3 inches, and that between the fifth and sixth months the birth weight is practically doubled and approximately trebled at the end of the twelfth month.



FIGS. 14 AND 15.—CHILD FIFTEEN MONTHS OLD.

Normal in every respect except 10 per cent over average weight. This can hardly be considered very abnormal at this age. The posture in Fig. 14 is normal for this age.

During the first twelve months, therefore, so far as height and weight are concerned, these statements may be considered as safe guides in judging growth and nutrition. In a breast-fed baby the weekly increase in weight should be between 5 and 6 ounces during the early



FIGS. 16, 17, AND 18.—THREE TYPES OF POSTURE AND CONTOUR REPRESENTING CHILDREN FROM ONE YEAR UP TO SIX YEARS.

They are composite diagrams compiled from a large number of children. There is relatively little change in posture from the first to the sixth year, and the contour changes only so far as slender, medium, and stocky individuals are concerned. Posture during these ages, as represented by these diagrams, differs considerably from the curves of the spine as portrayed in Fig. 5, yet since the diagrams were drawn from life and compared with actual photographs, the author feels that these diagrams more nearly represent the facts, as observed in the average child. The accompanying photographs are chosen as those of children showing average postures, little regard being paid to nutrition as judged by the relation of weight to height.

The influence of sex on posture or contour is not so noticeable as in older children. It will also be noted that the chest has not expanded sufficiently to make it protrude beyond the chin line, and that the abdomen is uniformly protuberant. Children during the years represented are, however, uniformly erect.

portion of the period of lactation. After the fourth month, 4 to 5 ounces per week is the usual gain in weight.

The normal breast-fed infant, securing a sufficient amount of mother's milk to cause it to gain at the rate just mentioned, is evidently satisfied after a ten-, or at the outside fifteen-minute nursing, and falls

asleep promptly on the completion of the meal. Such an infant sleeps from twenty to twenty-two hours each day during the early months of life, while during the later months of the first year from sixteen to eighteen hours are a normal average. The normal breast-fed infant does not cry except just before the hour of nursing, and some infants not then, and at the time of the daily bath. There is one notable exception: some mothers supply an ample amount of milk at the first two or three nursings in the day but at the later nursings do not supply enough to satisfy the appetite of the child. In these cases, the child cries after one or more of the later feedings and this is often mistaken for colic. Many of these children gain at the regular rate per week because



FIG. 19.—REPRESENTING POSTURE SHOWN IN FIG. 16.

they secure a sufficient amount during the early feedings of the day on which to gain, the deficiency at the later feedings apparently causing no disturbance of nutrition and only an annoying spell of crying. Routine observations by weighing before and after each nursing will readily establish the facts. When a normal breast-fed infant cries at other times than those just mentioned, the cause is apt to be discomfort from wet or too tightly applied diapers; from being overclothed with consequent overheating, or from irritating clothing, or from being spoiled. The last cause can be proven readily by taking the child up when it cries, and if the crying ceases within a few moments, it is most likely spoiled.



occurring from time to time in average measurements, at one time in favor of the head, and at another time in favor of the chest.

**Weight.**—We have seen that during the first three-year period of life the uniformity of growth among all children is quite constant, as is the increase in weight under *normal feeding conditions*, so that the relation of weight to age is a fairly satisfactory guide during this period



FIG. 22.—FRONT, SIDE, AND REAR VIEW OF CHILD AGED TWO YEARS, THIRTY-SEVEN INCHES TALL, WEIGHING THIRTY POUNDS.  
Slightly underweight but in other respects normal.

and a weight curve in an individual child which parallels curves based on averages and considered as normal, means more than the actual gain during this period. It will be found that not infrequently an infant weighing much less than the average at birth, and especially one who is premature, will often reach the average weight at one year, whereas an infant weighing 8 or 9 pounds at birth will weigh no more than the average at the end of twelve months. There would appear, therefore, to be a law of compensation which acts in some unknown manner. It is quite likely that a premature, underweight child increases in weight at a more rapid rate because of an unusual amount of attention being

paid it; and it must be remembered that the growth impulse is greatest in the premature. Such infants can in no sense be considered normal when they are born, but when they have reached the average for their age or height, we frequently find that they meet all requirements for normal children; therefore in this class we must frequently ignore the past history in determining whether the child is normal or not.

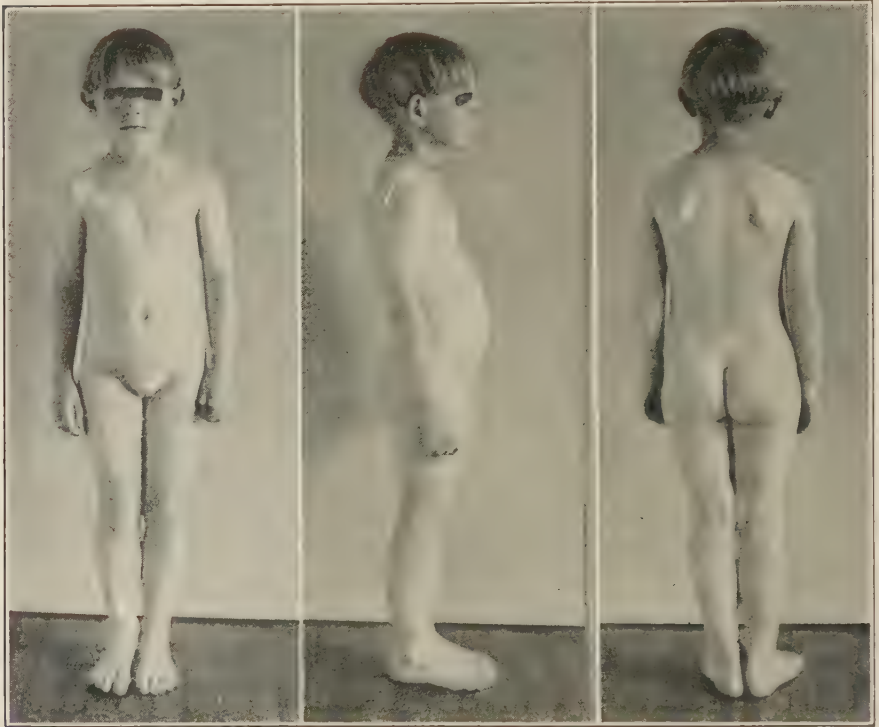


FIG. 23.—FRONT, SIDE, AND REAR VIEWS OF CHILD THIRTY-EIGHT AND ONE-HALF INCHES TALL, WEIGHT THIRTY-THREE POUNDS FOURTEEN OUNCES.

Posture nearly normal for age (three and one-half years). Legs show evidence of old rickets. Tuberculin test positive, but no demonstrable lesion.

Apparent lateral curvature of spine due to position before the camera.

It is a common experience to find a breast-fed infant weighing from 15 to 25 per cent more than the average, and although it is decidedly not a normal child, we are unable at present to state to what extent it is abnormal. In artificially fed children the feeding history in markedly overweight children will often show that an excessive amount of carbohydrate has been used in the food. During the second and third years also we find a considerable number of children who are over-

weight in whom no other abnormality will be demonstrated. A weight during this period markedly in excess of the average must be considered abnormal. Of more importance, however, than a slight variation on either side of the weight curve is the firmness of the fat or subcutaneous



FIG. 24.—FRONT, SIDE, AND REAR VIEWS OF FOUR-YEAR-OLD CHILD, THIRTY-SEVEN AND THREE-FOURTHS INCHES TALL, WEIGHT THIRTY-FOUR AND ONE-HALF POUNDS.

Normal child of stocky type.

turgor. Only experience will enable one to appraise this sign properly. The skin of a normal child is smooth, velvety and moist. In certain instances in obese children and sometimes in those who apparently have a normal amount of subcutaneous fat, there is an irregular feeling in passing the hand lightly over the skin and a certain brawny consistency to the fat, which is evidently abnormal, but the cause of it is not known. It has been attributed by some observers to a disturbance in the endocrine system. The distribution of fat is uniform and

whereas in older children we consider deposits in unusual localities, such as the upper pelvic girdle, over the sacral triangle, and in the region of the mammae and shoulders, as distinctly abnormal and possibly suggestive of endocrine disturbance, it is not so considered in children



FIG. 25.—REAR, FRONT, AND SIDE VIEWS OF CHILD SIX AND ONE-HALF YEARS OLD, FORTY-NINE AND ONE-FOURTH INCHES TALL, WEIGHING FIFTY-SEVEN AND ONE-HALF POUNDS.

This child retains the childish posture but approaches the adult contour to an extent rarely seen at this age.

of the age under consideration; in fact, in moderate amounts, such distribution of fat is almost uniform in a well-nourished child.

**Posture.**—In older children, posture is one of the best criteria of the normal state of nutrition. In children during the first three years of life, the posture is fairly uniform. Malnutrition has relatively slight



following fairly closely the three well-recognized racial types—hypomorph, mesomorph, and hypermorph. During the first three years the child has a distinctly hypomorph posture and contour, which shows a resemblance to the posture of primitive man, and which endures in some of the yellow and yellow-brown races of to-day. At this age the cervical curve of the spine is marked, throwing the head normally farther forward. The lumbar spine has a decided anterior convexity, resulting in a quite normal lordosis. It is likely that these characteristics are due to the gradual assumption of the erect position, and the gradual development of the muscles incident to this change. From three years to seven or eight years the posture becomes more of the mesomorph type; the cervical spine and the lumbar region straightening out somewhat, so that the head is held more erect than at the earlier period and the lumbar spine becomes less lordotic. This is closely allied to the mesomorph type. After the seventh or eighth years the normal adult posture is acquired. The relation between the length of the torso and the lower extremities gradually becomes that of the normal adult. It will be seen therefore that the usual charts representing normal stature refer only to the older child and not to the younger child. An effort has been made to illustrate these differences by means of the accompanying diagrams and photographs of children actually observed.

It has already been stated that a thorough physical examination is presupposed when we attempt to determine the state of nutrition. A few points must be stressed in order that we may have a full appreciation of their relation to the state of nutrition. A syphilitic child, although showing no marked clinical evidence of the disease, cannot be considered normal



FIG. 29.—DIAGRAM OF EXCELLENT POSTURE OF GIRLS OVER SIX YEARS OF AGE.

of the one into the other. We have considered the first three years separately because there is quite a marked difference during those years from later childhood. The differences from that time on throughout childhood and youth are not so marked, and these periods will be considered together.



FIG. 32.—FRONT, REAR, AND SIDE VIEWS OF ELEVEN-YEAR-OLD GIRL, SIXTY AND ONE-FOURTH INCHES TALL, WEIGHING NINETY AND THREE-FOURTHS POUNDS.

Was first seen one month prior to photograph, for suspicious hilus tuberculosis and suffering from otitis media, and chronic tonsillitis. She has gained a little over two pounds since first seen. The infections have had little or no effect on her posture, which represents fairly well the "good" posture. In the photograph she is tilted a little too forward, and when erect the arms hang farther back.

The general appearance of the child should be considered first. The "general appearance" includes facial expression, whether bright, intelligent and alert; the adenoid facies, or a somewhat similar expression due to a high palatine arch, or to malocclusion of the teeth. The color of skin and mucous membranes, though not always to be determined by mere inspection and often a misleading observation, should aid to some extent in our first impression as to normality. The complexion (whether blond or brunette) must be considered, since each has its own significance as to what may be considered a "healthy complexion." The skin of a normal healthy child is moist, of a velvety feel, and a certain clearness or freedom from blotching, pallor or anything resembling cachexia. Deviations from the normal are often apparent on the most cursory inspection.

Muscle tone in the normal child is not to be determined by any superficial examination. Dynamic implements are required as in the adult, if extreme accuracy is desired, but this is rarely practicable, and in fact, few if any observations have been made by which we might compare children of the same age, or the same child at different ages. A certain firmness of the muscle groups is recognized by ordinary palpation and pressure, while promptness of response in directed actions such as jumping, running, bending, and the like indicate both tone and coördination of the muscles. Agility and coördination of muscle action, easily determined by simple tests such as the above or others which may be suggested by the examiner, not only show the actual present condition of the child, but also serve to determine what the state of nutrition in the past has been, and frequently indicate whether a former state of ill health has been of brief or prolonged duration. A fair index of the state of the nervous system may be gained by testing certain reflexes, particularly those elicited by means of the percussion hammer. The mental development is often indicated by the same tests, since a child whose mentality is retarded is notably slow in muscle response and especially in coördination. Individual peculiarities in all these matters are marked and no final conclusion should be arrived at in doubtful cases until repeated observations at different and widely separated intervals have been made.

Subcutaneous fat is uniformly distributed, although in very young children, and even in some older ones, fat pads are present in the upper pelvic girdle and over the sacrum. These pads differ in size in different individuals, and although they bear some relation to the amount of subcutaneous fat, are prominent in some relatively spare

children and hardly noticeable in some who tend to the obese type. In our present state of knowledge their significance is not established.

In older children, besides the characteristics just reviewed, there are four main criteria by which we judge development and the state of nutrition, and thereby determine normality. They are: weight for age, height for age, and weight for height, and posture. Weight for age as we have seen is not a satisfactory guide, but is often used with a fair degree of accuracy in children before the age of three; but even at that early age, if the child is taller than the average for its age, it is better to use weight for height as a guide. Height in relation to age gives an approximate idea of the development of the bony skeleton only, that is stature, and varies so markedly in different individuals as to be often misleading. We are again confronted with the question of whether we are to take standards based on an average of a large number of children from all walks of life, and from localities in which there is a generous amount of racial admixture such as are found in the public schools of large communities, or whether our standards should be based on children attending private schools in localities where the economic and social status is high, and where the stock is largely if not entirely Anglo-Saxon. Studies have been made on both classes of children and the results differ materially. The children of the private-school class are taller and heavier for their age than those of the public-school class; on the other hand the public-school children show an advantage in weight for height. It has been decided to take the average from public-school children as our normal, as this will serve as a safer guide to the greatest number of observers. In average children a fairly definite ratio exists between weight and height. No one can say that a child must weigh exactly a certain number of pounds for a certain number of inches of height in order to be considered normal from a nutritional standpoint. There is probably a degree of latitude to be allowed on either side of the average line, and most observers agree that 7 per cent is a safe margin. For this purpose a table showing such averages is convenient for rapid reference. The tables shown here are compilations of averages from a large number of examinations by different observers, in widely separated parts of the country.



# THE NORMAL CHILD

III

TABLE OF AVERAGE WEIGHTS OF BOYS AT VARIOUS HEIGHTS \*

*Also Showing Weights 7 Per Cent Underweight for Height*

Height, Inches †	Average Weight for Height, Pounds	7 Per Cent Underweight, Pounds	Height, Inches †	Average Weight for Height, Pounds	7 Per Cent Underweight, Pounds
20.00	8.0	7.5	34.00	27.0	25.0
20.25	8.5	8.0	34.25	27.5	25.5
20.50	9.0	8.5	34.50	28.0	26.0
20.75	9.5	9.0	34.75	28.5	26.5
21.00	9.5	9.0	35.00	29.5	27.5
21.25	9.5	9.0	35.25	30.0	28.0
21.50	10.0	9.5	35.50	30.5	28.5
21.75	10.5	10.0	35.75	31.0	29.0
22.00	10.5	10.0	36.00	31.0	29.0
22.25	11.0	10.0	36.25	31.5	29.5
22.50	11.5	11.0	36.50	32.0	30.0
22.75	12.0	11.0	36.75	32.0	30.0
23.00	12.0	11.0	37.00	32.0	30.0
23.25	12.5	11.5	37.25	32.5	30.0
23.50	13.0	12.0	37.50	33.0	30.5
23.75	13.5	12.5	37.75	33.5	31.0
24.00	13.5	12.5	38.00	34.0	31.5
24.25	14.0	13.0	38.25	34.5	32.0
24.50	14.5	13.5	38.50	35.0	32.5
24.75	15.0	14.0	38.75	35.0	32.5
25.00	15.0	14.0	39.00	35.0	32.5
25.25	15.5	14.5	39.25	35.5	33.0
25.50	16.0	15.0	39.50	36.0	33.5
25.75	16.5	15.5	39.75	36.0	33.5
26.00	16.5	15.5	40.00	36.0	33.5
26.25	17.0	16.0	40.25	36.5	34.0
26.50	17.5	16.5	40.50	37.0	34.5
26.75	18.0	16.5	40.75	37.5	35.0
27.00	18.0	16.5	41.00	38.0	35.5
27.25	18.5	17.0	41.25	38.5	36.0
27.50	19.0	17.5	41.50	39.0	36.5
27.75	19.5	18.0	41.75	39.0	36.5
28.00	19.5	18.0	42.00	39.0	36.5
28.25	19.5	18.0	42.25	39.5	36.5
28.50	20.0	18.5	42.50	40.0	37.0
28.75	20.5	19.0	42.75	40.5	37.5
29.00	20.5	19.0	43.00	41.0	38.0
29.25	21.0	19.5	43.25	42.0	39.0
29.50	21.5	20.0	43.50	43.0	40.0
29.75	22.0	20.5	43.75	43.5	40.5
30.00	22.0	20.5	44.00	44.0	41.0
30.25	22.0	20.5	44.25	44.5	41.5
30.50	22.5	21.0	44.50	45.0	42.0
30.75	23.0	21.5	44.75	45.5	42.5
31.00	23.0	21.5	45.00	46.0	43.0
31.25	23.5	22.0	45.25	46.5	43.5
31.50	24.0	22.5	45.50	47.0	43.5
31.75	24.5	23.0	45.75	47.5	44.0
32.00	24.5	23.0	46.00	48.0	44.5
32.25	25.0	23.5	46.25	48.5	45.0
32.50	25.5	23.5	46.50	49.0	45.5
32.75	26.0	24.0	46.75	49.5	46.0
33.00	26.0	24.0	47.00	50.0	46.5
33.25	26.0	24.0	47.25	51.0	47.5
33.50	26.5	24.5	47.50	52.0	48.5
33.75	27.0	25.0	47.75	52.5	49.0

\* From the Literature of the Nutrition Clinics for Delicate Children, Boston, W. R. P. Emerson, M.D., Director.

† Without clothing.

## THE NORMAL CHILD

TABLE OF AVERAGE WEIGHTS OF BOYS AT VARIOUS HEIGHTS \*

*Also Showing Weights 7 Per Cent Underweight for Height*

Height, Inches †	Average Weight for Height, Pounds	7 Per Cent Underweight, Pounds	Height, Inches †	Average Weight for Height, Pounds	7 Per Cent Underweight, Pounds
48.00	53.0	49.5	62.00	104.0	96.5
48.25	53.5	50.0	62.25	106.0	98.5
48.50	54.0	50.0	62.50	108.0	100.5
48.75	54.5	50.5	62.75	110.0	102.5
49.00	55.0	51.0	63.00	111.0	103.0
49.25	56.0	52.0	63.25	112.5	104.5
49.50	57.0	53.0	63.50	114.0	106.0
49.75	57.5	53.5	63.75	115.5	107.5
50.00	58.0	54.0	64.00	117.0	109.0
50.25	59.0	55.0	64.25	118.5	110.0
50.50	60.0	56.0	64.50	120.0	111.5
50.75	60.5	56.0	64.75	121.5	113.0
51.00	61.0	56.5	65.00	123.0	114.5
51.25	62.0	57.5	65.25	124.5	116.0
51.50	63.0	58.5	65.50	126.0	117.0
51.75	63.5	59.0	65.75	127.5	118.5
52.00	64.0	59.5	66.00	129.0	120.0
52.25	65.0	60.5	66.25	130.0	121.0
52.50	66.0	61.5	66.50	131.0	122.0
52.75	67.0	62.5	66.75	132.0	123.0
53.00	68.0	63.0	67.00	133.0	123.5
53.25	69.0	64.0	67.25	134.5	125.0
53.50	70.0	65.0	67.50	136.0	126.5
53.75	70.5	65.5	67.75	137.5	128.0
54.00	71.0	66.0	68.00	139.0	129.5
54.25	72.0	67.0	68.25	140.5	130.5
54.50	73.0	68.0	68.50	141.5	131.5
54.75	73.5	68.5	68.75	143.0	133.0
55.00	74.0	69.0	69.00	144.0	134.0
55.25	75.0	69.5	69.25	145.0	135.0
55.50	76.0	70.5	69.50	146.0	136.0
55.75	77.0	71.5	69.75	146.5	136.5
56.00	78.0	72.5	70.00	147.0	136.5
56.25	79.0	73.5	70.25	148.5	138.0
56.50	80.0	74.5	70.50	149.5	139.0
56.75	81.0	75.5	70.75	151.0	140.5
57.00	82.0	76.5	71.00	152.0	141.5
57.25	83.0	77.0	71.25	153.5	143.0
57.50	84.0	78.0	71.50	154.5	143.5
57.75	84.5	78.5	71.75	156.0	145.0
58.00	85.0	79.0	72.00	157.0	146.0
58.25	86.0	80.0	72.25	158.5	147.5
58.50	87.0	81.0	72.50	160.0	149.0
58.75	88.0	82.0	72.75	161.5	150.0
59.00	89.0	83.0	73.00	163.0	151.5
59.25	90.5	84.0	73.25	164.5	153.0
59.50	91.5	85.0	73.50	166.0	154.5
59.75	93.0	86.5	73.75	167.5	156.0
60.00	94.0	87.5	74.00	169.0	157.0
60.25	95.5	89.0			
60.50	96.5	90.0			
60.75	98.0	91.0			
61.00	99.0	92.0			
61.25	100.5	93.5			
61.50	101.5	94.5			
61.75	103.0	96.0			

\* From the Literature of the Nutrition Clinics for Delicate Children, Boston, W. R. P. Emerson, M.D., Director.

† Without clothing.

TABLE OF AVERAGE WEIGHTS OF GIRLS AT VARIOUS HEIGHTS \*

*Also Showing Weights 7 Per Cent Underweight for Height*

Height, Inches †	Average Weight for Height, Pounds	7 Per Cent Underweight, Pounds	Height, Inches †	Average Weight for Height, Pounds	7 Per Cent Underweight, Pounds
20.00	8.0	7.5	34.00 †	26.5	24.5
20.25	8.0	7.5	34.25 †	27.0	25.0
20.50	8.5	8.0	34.50 †	27.0	25.0
20.75	9.0	8.5	34.75 †	27.5	25.5
21.00	9.0	8.5	35.00	29.0	27.0
21.25	9.5	9.0	35.25	29.0	27.0
21.50	10.0	9.5	35.50	29.5	27.5
21.75	10.5	10.0	35.75	30.0	28.0
22.00	10.5	10.0	36.00	30.0	28.0
22.25	11.0	10.0	36.25	30.5	28.5
22.50	11.5	11.0	36.50	31.0	29.0
22.75	12.0	11.0	36.75	31.5	29.5
23.00	12.0	11.0	37.00	31.5	29.5
23.25	12.5	11.5	37.25	32.0	30.0
23.50	13.0	12.0	37.50	32.5	30.0
23.75	13.5	12.5	37.75	33.0	30.5
24.00	13.5	12.5	38.00	33.0	30.5
24.25	14.0	13.0	38.25	33.0	30.5
24.50	14.5	13.5	38.50	33.5	31.0
24.75	15.0	14.0	38.75	34.0	31.5
25.00	15.0	14.0	39.00	34.0	31.5
25.25	15.5	14.5	39.25	34.5	32.0
25.50	16.0	15.0	39.50	35.0	32.5
25.75	16.5	15.5	39.75	35.5	33.0
26.00	16.5	15.5	40.00	36.0	33.5
26.25	16.5	15.5	40.25	36.0	33.5
26.50	17.0	16.0	40.50	36.5	34.0
26.75	17.5	16.5	40.75	37.0	34.5
27.00	17.5	16.5	41.00	37.0	34.5
27.25	18.0	16.5	41.25	37.5	35.0
27.50	18.5	17.0	41.50	38.0	35.5
27.75	19.0	17.5	41.75	38.5	36.0
28.00	19.0	17.5	42.00	39.0	36.5
28.25	19.0	18.0	42.25	39.5	36.5
28.50	19.5	18.0	42.50	40.0	37.0
28.75	20.0	18.5	42.75	40.5	37.5
29.00	20.0	18.5	43.00	41.0	38.0
29.25	20.5	19.0	43.25	41.0	38.0
29.50	21.0	19.5	43.50	41.5	38.5
29.75	21.5	20.0	43.75	42.0	39.0
30.00	21.5	20.0	44.00	42.0	39.0
30.25	21.5	20.0	44.25	43.0	40.0
30.50	22.0	20.5	44.50	44.0	41.0
30.75	22.5	21.0	44.75	44.5	41.5
31.00	22.5	21.0	45.00	45.0	42.0
31.25	23.0	21.5	45.25	45.5	42.5
31.50	23.5	22.0	45.50	46.0	43.0
31.75	24.0	22.5	45.75	46.5	43.5
32.00	24.0	22.5	46.00	47.0	43.5
32.25	24.0	22.5	46.25	48.0	44.5
32.50	24.5	23.0	46.50	49.0	45.5
32.75	25.0	23.5	46.75	49.5	46.0
33.00	25.0	23.5	47.00	50.0	46.5
33.25	25.5	23.5	47.25	50.5	47.0
33.50	26.0	24.0	47.50	51.0	47.5
33.75	26.5	24.5	47.75	51.5	48.0

\* From the Literature of the Nutrition Clinics for Delicate Children, Boston, W. R. P. Emerson, M.D., Director.

† Without clothing.

TABLE OF AVERAGE WEIGHTS OF GIRLS AT VARIOUS HEIGHTS \*

*Also Showing Weights 7 Per Cent Underweight for Height*

Height, Inches †	Average Weight for Height, Pounds	7 Per Cent Underweight, Pounds	Height, Inches †	Average Weight for Height, Pounds	7 Per Cent Underweight, Pounds
48.00	52.0	48.5	60.00	101.0	93.5
48.25	53.0	49.5	60.25	103.0	96.0
48.50	54.0	50.0	60.50	105.0	98.0
48.75	54.5	50.5	60.75	107.0	99.5
49.00	55.0	51.0	61.00	108.0	100.5
49.25	56.0	52.0	61.25	109.5	101.5
49.50	57.0	53.0	61.50	111.0	103.5
49.75	57.5	53.5	61.75	112.5	104.5
50.00	58.0	54.0	62.00	114.0	106.0
50.25	59.0	55.0	62.25	115.0	107.0
50.50	60.0	56.0	62.50	116.0	108.0
50.75	60.5	56.0	62.75	117.0	109.0
51.00	61.0	56.5	63.00	118.0	109.5
51.25	62.0	57.5	63.25	118.5	110.0
51.50	63.0	58.5	63.50	119.0	110.5
51.75	63.5	59.0	63.75	120.0	111.5
52.00	64.0	59.5	64.00	121.0	112.5
52.25	65.0	60.5	64.25	122	113.5
52.50	66.0	61.5	64.50	123.0	114.5
52.75	67.0	62.5	64.75	124.0	115.5
53.00	68.0	63.0	65.00	125.0	116.0
53.25	69.0	64.0	65.25	126.0	117.0
53.50	70.0	65.0	65.50	127.0	118.0
53.75	70.5	65.5	65.75	128.0	119.0
54.00	71.0	66.0	66.00	129.0	120.0
54.25	72.0	67.0	66.25	130.0	121.0
54.50	73.0	68.0	66.50	131.0	122.0
54.75	74.0	69.0	66.75	132.0	123.0
55.00	75.0	69.5	67.00	133.0	123.5
55.25	76.0	70.5	67.25	134.5	125.0
55.50	77.0	71.5	67.50	135.5	126.0
55.75	78.0	72.5	67.75	137.0	127.5
56.00	79.0	73.5	68.00	138.0	128.5
56.25	80.0	74.5	68.25	139.0	129.5
56.50	81.5	76.0	68.50	140.0	130.0
56.75	83.0	77.0	68.75	141.0	131.0
57.00	84.0	78.0	69.00	142.0	132.0
57.25	85.0	79.0	69.25	142.5	132.5
57.50	86.5	80.5	69.50	143.0	133.0
57.75	88.0	82.0	69.75	143.5	133.5
58.00	89.0	83.0	70.00	144.0	134.0
58.25	90.5	84.0	70.25	144.0	134.0
58.50	92.0	85.5	70.50	144.5	134.5
58.75	93.5	87.0	70.75	145.0	135.0
59.00	95.0	88.5	71.00	145.0	135.0
59.25	96.5	90.0			
59.50	98.0	91.0			
59.75	99.5	92.5			

\* From the Literature of the Nutrition Clinics for Delicate Children, Boston, W. R. P. Emerson, M.D., Director.  
† Without clothing.

Measurement of height should always be made without shoes, and weight preferably without clothing. It is obvious that under certain circumstances this is not feasible. A girdle cloth or bloomers may be used and practically no allowance need be made for the weight of



either of these. Where the child is weighed with clothing an allowance should be made for the clothes, which differ in different localities and at different seasons of the year in the same locality. A safe allowance for the ordinary clothing for a child of seven years or over is about 2 pounds.

The fourth of the criteria of normality alluded to is the posture. In the older child the posture closely approximates that of the mature adult. During the more advanced years of childhood and youth, the published diagrams furnish safe guides. At this time the individual is distinctly hypermorph. There is, however, no sharp line so far as age or even height are concerned at which the individual should belong to one or another of these types.

Development is gradual and form and stature do not appear suddenly, nor do individuals change over night from one type to another; so that in judging the normality of an individual one must exercise great care in arriving at a decision. Judgment of the examiner must ultimately determine the case, and this judgment can be acquired only after repeated examinations of many children and the learning to visualize normality rather than the following of a rule of thumb. Children are placed in three classes for this purpose: the slender type, the intermediate type, and the obese type. The *posture* is the same for each type; the contour differs markedly. The posture of the child usually considered normal will be described and illustrated here, and the abnormal posture considered in Chapter X on Malnutrition. The Federal Children's Bureau has issued a series of posture charts which illustrate in an excellent manner the normal posture of the *older child*, with deviations from the normal, and these diagrams are used here. They are based to a large extent on the previous work of Talbot and Brown (*Am. J. Dis. Child.*, 20: 168). The posture charts here presented for the younger child are original with the author, and were prepared from studies of a large number of children. It will be noted that three well-defined types are found among apparently normal young children. The first of the diagrams for older children (Fig. 26) is the normal posture, and is called excellent. The most noticeable feature of this posture is the erectness. The head is well poised and is carried high. It is so balanced above the shoulders, hips and ankles, that a plummet held at the external auditory meatus will pass through the center of the shoulder joint, the center of the hip joint, the center of the knee, and the center of the ankle. The head is so held that the chin is drawn in, and when this is viewed in relation to the well de-

veloped and expanded chest, it will be seen that the chest is outside of the chin line. The lower abdomen is flat and *within* the line of the chest. The spinal curves are definitely normal so that there is no bending of the neck forward, the shoulders have no tendency to "rounding"; the lumbar spine shows not the slightest tendency to lordosis. The arms hang so that the bend of the elbow is just about on a line with the lumbar curve. Relatively few children have such a perfect posture as this, but it is to be admired and sought after.

The second diagram (Fig. 27) shows a posture which is more frequently seen. It is considered good, but cannot be regarded as strictly normal. Corrections for this posture are, however, seldom advised. The most noticeable characteristic of this posture is that the whole body, instead of being perfectly erect as in the first diagram, is tilted slightly forward, apparently from the ankle. A closer study will explain the mechanics of the tilting. The cervical spine is bent forward, and the shoulders are beginning to assume a rounded appearance. The chest is not held so high and the chin is *on a line with the chest*. The lumbar spine is just a little more curved than in the first posture, but this is sufficient to throw the abdomen forward and on a line with the chest. The arm still hangs so that the bend of the elbow is not noticeably out of line with the lumbar curve.

The young child has a posture which is of a markedly different type from that of the older child; in fact closely resembles the posture of an older child which has acquired the "fatigue posture." Seldom do we see young children with a posture of the type described as normal in most of the literature. No sharp age line can be drawn at which each posture must be our guide. An effort has been made to describe and illustrate the postures of each age period by diagrams and photographs selected from a large number taken.

## CHAPTER VIII

### FOOD FOR THE NORMAL CHILD

The subject of nutrition in general is dealt with in this volume; the feeding of infants is considered in a separate volume, yet it will be necessary for the sake of continuity to explain here how the needs of the young child are to be met before taking up the feeding of the older child and the youth.

The food of a breast-fed infant is so well balanced by nature that no attention need be paid to it except occasionally to ascertain whether or not the infant is securing from the mother an amount sufficient for its needs. A steady gain in weight of 5 or 6 ounces each week during the early months of life attests an ample supply. A weekly gain of 4 ounces during the later months of breast feeding demonstrates the same fact. The quality of the food derived from the mother, with rare exceptions, is good, and the balance of the necessary food elements and accessory factors is so well regulated by nature that the infant develops normally, grows in weight in accordance with a steady curve, which as charted from a large number of observations we call normal, and is secure from nutritional disturbances and deficiency diseases. A daily record of the weight of a nursing infant will show frequent deviations from a steady curve. This is common and cannot be always accounted for. The supply of mother's milk is influenced by a number of circumstances, and in many instances the difference in the daily intake of water alone will determine whether or not the child gains in weight on a single day. A weekly weighing is not only usually sufficient but, in the home at least, is more desirable than a daily record. The weekly weight curve therefore should be our guide and should at least parallel the normal curve. When in doubt as to the amount of milk secured by the nursing infant, the simple expedient of weighing before and after each nursing will give the desired information. Generally speaking an average for the twenty-four hours of 2 ounces for each pound of body weight will promote a steady and sufficient gain in weight.

When an infant has to be weaned, before the usual time, or must be fed artificially from birth, a somewhat more serious and difficult problem presents. If, however, intelligent care is exercised in the very

beginning and our knowledge of nutrition is fairly well systematized, little or no difficulty will be found in producing quite satisfactory results. We have seen that a normal infant requires 1.5 grams of protein per kilogram of weight per day, to meet the requirements of waste and growth, and that this requirement is met by administering  $1\frac{1}{2}$  ounces of cows' milk per pound of weight per day. The fluid requirement of an infant is more or less definitely determined as 3 ounces per pound of body weight in twenty-four hours during the early months of life and approximately 2 ounces per pound per day during the later months of the first year. This fluid requirement of course includes the milk. This gives a fairly definite basis for the formula for a healthy infant. Cows' milk is deficient in carbohydrate for the needs of the infant even when undiluted; when diluted this deficiency is greater. It is necessary therefore to add some form of carbohydrate to the formula, usually one of the sugars. In many instances cane or ordinary table sugar will be found suitable, and has the further advantage of being cheaper. Lactose or milk sugar is rarely used to-day; the other alternative being one of the malt sugar and dextrin mixtures, dextrimaltose being a very desirable preparation. Malt sugar has the advantage of going through fewer changes in digestion to reach the dextrose stage, the form in which carbohydrate is absorbed. A fairly safe rule in the administration of sugar is that for an infant under 10 pounds in weight, 1 ounce of sugar should be added to the cows' milk formula and for an infant weighing over 10 pounds  $1\frac{1}{2}$  ounces should be added. Every formula should be checked by the caloric requirements of the individual infant. The requirements of total food and of the various food elements may be found in Chapter X.

This method of feeding the normal infant by means of simple dilutions of whole milk gives quite satisfactory results in the majority of cases. As the infant nears the close of the first year whole milk may be substituted for the formula of diluted milk.

For a long time during the advance of knowledge of nutritional requirements of the infant and growing child, although the relative importance of protein was recognized, its full significance was not understood, and at the same time protein was regarded as the most difficult of the elements for the infant to digest. The result of this opinion was that formulæ were so made that the protein was reduced to a minimum. Gradually it was shown that protein was not so difficult of digestion as at first thought. It was not known why protein was a tax on the digestive system, but recent investigations have proven



that the real and only difficulty arose from the fact that certain substances bound such a large proportion of the hydrochloric acid of the gastric juice that very little was left to perform other important functions. These substances are the phosphates and caseinates, and are known as the buffer substances. It was also found that when these substances were neutralized before ingestion, by acidifying the milk, there was no difficulty in the digestion of the protein. The degree of acidity or hydrogen-ion concentration ( $P^H$ ) of the gastric contents of the infant at the height of the digestion of human milk, was found to be 3.75, and this was considered optimum. Cows' milk was then acidified to the extent of 3.75 before ingestion and it was found that very young infants, even premature infants, could digest undiluted cows' milk without difficulty. A number of different acids were tried, but finally it was proven by Marriott and his coworkers that lactic acid served the purpose better than other acids. This discovery has been a great addition to our knowledge of infant feeding and has simplified this difficult problem considerably. In cows' milk the principal deficiency compared with human milk is in the carbohydrate, and this therefore must be added as stated above. In the preparation of lactic acid milk, the acid should be added in the proportion of 1 dram of acid to 1 pint of milk. The milk is boiled and then chilled and the acid added drop by drop, the milk being stirred constantly, until the mixture is smooth and homogeneous. This requires about twenty minutes. It is important that the exact proportions be maintained by accurate measurement and not by guess. Only in this way can proper acidification be secured. To the acidified milk some form of sugar is added.

Marriott feels that corn syrup is the most easily assimilated form in which to give sugar; other observers, however, including the University of Virginia Hospital, feel that dextrimaltose, from a practical standpoint at least, serves the same purpose. The infant is fed on this mixture in accordance with its caloric needs. The lactic-acid milk contains about twenty calories to the ounce, and sugar about one hundred and twenty to the ounce.

Not only the caloric requirements of an infant may be met through the use of lactic-acid milk with the added sugar, but the protein requirements are met also. In many instances infants are fed protein in excess of their needs and often far in excess, apparently with no ill effect. Lactic-acid milk has been in use for a rather short time, and it may yet be found that some finer adjustment to the digestive capacity may be necessary.

The above descriptions of the feeding of a normal infant are for the period of exclusive milk feeding. The question of when to give other solid or semisolid foods, cereals, vegetables, and fruits is answered in almost as many ways as there are those who attempt to regulate and prescribe the food of infants.

Although orange juice is given primarily for the prevention of scurvy when an infant is fed on other than human milk, especially on boiled cows' milk, it is a safe procedure to begin all children on a daily administration of orange juice about the third month.

Sometimes a cessation of growth occurs at or near the sixth month, even when there is apparently adequate food. Some observers consider this a manifestation of latent scurvy, and therefore it would seem advisable to use orange juice as a routine, on account of its vitamin C content which is antiscorbutic as well as growth-promoting. When orange juice is not available, the juice of the tomato may be substituted. It is well to begin the weaning from the breast about the sixth month by the substitution of one artificial feeding for one breast feeding each day and then two and so on, so that in the course of from two to three weeks the child is entirely on the bottle or cup. Whether a child is on the breast entirely, or on part breast and part bottle, or entirely on artificial feeding, it is wise to begin the administration of food other than milk at or near the sixth month. Fresh vegetables in the form of broth or purée may be given in small quantities at first and gradually increased in amount. Spinach, turnip tops, kale and other leafy vegetables may be cooked, preferably by steaming rather than boiling, cut up fine, forced through a sieve and added to a meat or vegetable broth, soup or purée. A very convenient preparation for this purpose is a cream, or clear vegetable soup, such as is put up in cans by several reliable commercial concerns. One-half teacup may be given in the beginning, and should be gradually increased until a cupful is given. At the same time bread, one day old, should be given once a day. After this cereals may be begun. Those of the wheat variety, such as farina, cream of wheat or wheatena, are quite suitable. The time of beginning egg as a part of the diet varies with circumstances, usually about the tenth month. It may be given once or twice a week in the beginning, and increased to every other day by the twelfth month. A familiar article of diet in the southern states is batter bread or spoon bread, made from corn meal, milk and eggs, and is in itself a fairly balanced meal for the older infant as well as for children. The ad-

visability of routine administration of cod-liver oil is discussed in Chapter XIII on Rickets.

Habits of eating are quite as important in preventing malnutrition as is the food itself. From a physiological standpoint there may be no objection to feeding irregularly, but from a practical standpoint in handling infants and children, habits of eating are of great importance. The most approved interval of feeding infants, and one which is well-nigh universally used by pediatricians, is the four-hour interval. For convenience in the home and especially in institutions, a six, ten, two, six, ten, two, feeding schedule will be found the most practical. As soon as an infant will go from ten at night to six in the morning without waking, the 2:00 A.M. feeding may be omitted. A number of good clinicians do not feed at two in the morning at all. This will do well for robust infants, but it is a doubtful policy for those who are below the average weight at birth. As a rule, however, the 2:00 A.M. feeding may be omitted by the second month. The 10:00 P.M. feeding may usually be omitted by the fifth or sixth month, without detriment.

At twelve months of age the child is still on a four-hour interval of feeding. A safe daily menu for an infant from the twelfth to the fourteenth month is as follows:

6 A.M. Whole milk from a cup. Usually 8 ounces will be sufficient.

8 A.M. Orange juice, or prune juice. These may be given at the ten o'clock or the two o'clock meal.

10 A.M. Milk (8 ounces which is often not entirely consumed if much solid food is taken at this meal). Two to four tablespoonfuls of farina, wheatena, or cream of wheat. One slice of crisp bacon.

2 P.M. One slice of one day old baker's bread. (Toast or Zwieback may be given, though they possess no advantage over the dry bread.) Broth as already described, and occasionally a little scraped beef or beef juice (drippings from roast or steak.) An egg may be given instead of the meat or meat juice. When broth or soup is not given milk should be, otherwise milk may be omitted at this meal.

6 P.M. Milk (8 oz.) Toast, or dry bread, or cereal, occasionally custard, and as a general routine some form of cooked fruit such as prunes or apple sauce.

Although milk is in itself an almost perfect food, and should constitute a large part of the diet of every growing child, it has its limitations, and should not be used exclusively during the latter part of the first year nor during the second year. Milk does not contain enough iron for the needs of a growing child during the periods just mentioned, and so to prevent the anemia so commonly observed at this time and known as "milk anemia," other foods are given, particularly the green vegetables.

From the fourteenth to the eighteenth month the food need not differ materially from the table just given. An increase in quantity is made gradually and other articles of the same general character are added. As the child takes more solid food the quantity of milk is gradually reduced, so that at the eighteenth month from a pint to a pint and a half is consumed each day in its natural state. Many prepared articles of diet are made up with milk and this should be counted in the total quantity. According to McCollum a balanced ration is provided in milk, eggs, leafy vegetables, and fresh fruits.

In feeding children of the age under discussion and also those still older, the prime consideration should be to insure a sufficient amount of protein each day to meet the requirements of waste and development. Next in importance is the total number of calories needed for increase in weight. Milk and eggs furnish all the protein needed, but for the sake of variety other protein foods are given in a manner to be described. All the fat needed for ordinary metabolic processes is secured in a well-balanced meal, and if more is deemed advisable, enough butter can be added to meet these requirements. After the protein needs are met in the arrangement of a dietary, a sufficient number of calories to make up the deficiency can be added in the form of carbohydrates, such as sugar, cereal, and bread.

At eighteen months the average normal child should be on a schedule of three meals a day. This implies a five-hour régime, and the most convenient hours for the average household are 8:00 A.M., 1:00 P.M., and 6:00 P.M. Nothing should be eaten by the normal child between meals, but an abundance of water should be drunk.

In arranging the daily menu for normal children, variety is not an important consideration so far as the needs of the body are concerned, but is quite important in maintaining a vigorous appetite, and this consideration becomes more and more important as the child grows older. Children should be taught to eat what is put before them and eat the food for the sake of the food alone; in other words, the desires of the child for certain articles of food should not be catered to for



fear of developing a capricious appetite. But varying the separate articles of food within justifiable limits has a tendency to keep alive a certain expectancy, with consequent appreciation of the food when it appears on the table as a surprise. The following list of food articles so grouped that interchangeable foods are recognized at a glance will be found serviceable. Following this list is another, containing a number of articles which are forbidden for some definite reason.

**Milk.**—Wherever possible, fresh cows' milk should be used. In infancy raw milk should not be given because we are never quite sure of its purity. After three years of age children can usually be safe in taking raw milk. Where fresh milk is not available one of the dried milk preparations, such as Klim or Dryco, may be used to advantage. Buttermilk may be allowed, but it must be remembered that in ordinary dairy buttermilk, the food value is only about one-half of that of the fresh product.

**Fruits.**—Give fresh apples, peaches, apricots, plums,<sup>1</sup> pears, oranges, grapefruit, berries <sup>1</sup> (in season), baked apples, stewed prunes, applesauce, baked pears, stewed pears, peaches and apricots. The dried and canned fruits serve a good purpose in localities where the fresh products are not available, or during the winter season. The vitamin and food content is impaired somewhat in the dried articles, but modern canning methods retain a large proportion of both. The factor of aging has not been studied sufficiently at present to justify an opinion.

**Cereals.**—Give oatmeal, cracked wheat, cream of wheat, farina, wheatena, samp,<sup>1</sup> rice and small hominy.

**Starchy Vegetables.**—Give white potatoes (baked slowly in the jacket), green peas, young butter beans.

**Leaf and Stem Vegetables.**—String beans, asparagus, lettuce, onions, beets, Swiss chard, squash, celery (stewed), beet greens, tomatoes, turnips, carrots, spinach, kale, turnip tops, and cabbage,<sup>2</sup> may be given.

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<sup>1</sup>Do not give to children under two years of age without the recommendation of a physician.

<sup>2</sup>Cooked cabbage may be given children of two years and over provided it is prepared in the following manner: Take off the outside leaves. Cut the cabbage in quarters and remove the tough stalk. Shred. Soak in cold water until crisp. Drop into an uncovered vessel with a very little boiling water (enough to cover bottom of the vessel) and, if fresh cabbage, cook for twenty minutes covered nearly but not quite tight. Cook until the water is gone but the cabbage still moist, or until it is tender. Serve with a cream sauce and with butter and salt. The length of time of cooking depends upon the age of the cabbage but it should be the least possible time to soften it. If it takes thirty minutes the cabbage is too old and tough for children.

**Bread.**—Loaf bread (one or two days old), whole wheat, graham, rye, corn or flour, crackers, made from all of the materials just mentioned, batter bread (corn bread, spoon bread or egg bread) are given.

**Desserts.**—Give junket, custard, tapioca, sago, bread or rice pudding, prune whip, stewed fruit, jelly made with gelatin, ice cream (vanilla, frozen custard or chocolate).

**Eggs, Fish, Meat.**—Eggs may be given cooked any way except fried. Hard boiled eggs are more easily digested than those cooked in any other way. Fresh fish such as cod, haddock, halibut, trout, spots, rock, or in fact any of the dry or non-oily fish. All fish should be boiled, broiled or baked, never fried. Chicken, turkey, lamb, roast beef. Meat should be given sparingly at all ages, since more valuable protein is furnished by milk and eggs.

**Soups.**—Cream, chicken or vegetable soups are suitable. Soups of the ordinary kind supply very little nutriment. Vegetable soups made by boiling several vegetables supply vitamins in abundance. Soups may be made more nourishing by the addition of milk, rice or barley.

**Fats.**—Butter,<sup>3</sup> bacon (fried crisp), olive oil, and peanut butter.

The following foods should not be a part of the diet of children :

1. Tea, coffee, soda water, beer, whiskey, wine, cider
2. Fried foods of any kind
3. Pork, veal, kidneys, greasy stews
4. Cucumbers, corn (unless very young and tender)
5. Fresh bread, hot bread, buns, pie, fried cakes, rich puddings
6. Nuts, raisins (except stewed), berries (only for older children)
7. Sweets; some authorities insist that no candy, cakes or sweet drinks should be given young children. All insist that if allowed, they should be given seldom, and then only at the end of the midday meal. They should never be allowed between meals, that is on an empty stomach.

It has been seen in a previous chapter that the daily requirements of protein, fat, and carbohydrate are known with a fair degree of accuracy. During the second year the protein needs approximate 44 grams per day. By the sixth or seventh year 60 grams per day are

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<sup>3</sup> Butter fat should be given a decided preference over other fats. Butter substitutes may be used where there is plenty of whole milk and fresh leaf and stem vegetables in the diet.

required; by the twelfth year 90 grams and from the twelfth to the fifteenth year the requirement is 130 grams per day. Translated into a quotient it will be seen that from the first to the sixth year the protein need is from 3 to 4 grams per kilogram of body weight per day, while after the sixth year it is 2 grams per kilogram per day.

Throughout late infancy and childhood the fat requirement is approximately 3 grams per kilogram per day. The carbohydrate needs also remain fairly constant after the first year of life and are 10 grams per kilogram per day. In older children this requirement is met by both soluble and insoluble carbohydrates, about one half from one and one half from the other.

Of the total calories needed for the average normal child 15 per cent should be supplied by protein; 35 per cent by fat and 50 per cent by carbohydrate.

According to Sherman the caloric requirements at different ages are:

<i>Age, Years</i>	<i>Calories per Day</i>
1- 2 .....	1000-1200
2- 5 .....	1200-1500
6- 9 .....	1400-2000
10-13 (girls) .....	1800-2400
10-13 (boys) .....	2300-3000
14-17 (girls) .....	2200-2600
14-17 (boys) .....	2800-4000

In terms of a quotient:

<i>Age, Years</i>	<i>Calories per Kilogram</i>	<i>Calories per Pound</i>
Under one year.....	100	45
1- 2 .....	100-90	45-40
2- 5 .....	90-80	40-36
6- 9 .....	80-70	36-32
10-13 .....	75-60	34-27
14-17 .....	65-50	30-22

Based on these facts an effort has been made to outline a series of diets to meet the needs of the several age groups for the average healthy child, to serve as guides in feeding normal children in such a manner that the requirements of development, growth, repair, activity and waste are met.

## A DAY'S DIETARY FOR A CHILD OF TWO YEARS

Approximate value for day's diet { 15 per cent Protein, 45 grams, 180 calories  
 35 per cent Fat, 46.6 grams, 420 calories  
 50 per cent Carbohydrate, 150 grams, 600 calories

Fuel value: 1200 calories

Food	Amount	Weight, Grams	Pro- tein, Grams	Fat, Grams	Carbo- hydrate, Grams	Cal- ories	Vitamins
<i>Breakfast: 8 A. M.</i>							
Orange .....	½ glass	4 oz.	.9	.24	12.8	60	B & C
Cream of wheat ....	¾ cup *	25	2.5	.2	18.3	85	A & B
Egg .....	1	57	6.8	5.3	....	75	A & B
Toast or stale bread	1 slice †	20	1.9	.18	10	50	A & B
Milk .....	Scant ⅝ glass	5 oz.	4.7	5.7	7.2	100	A, B & C
TOTAL .....			16.8	11.62	48.3	370	
<i>Dinner: 1 P. M.</i>							
Baked potato .....	1 medium	150	2.7	.15	22	100	B & C
Spinach (chopped) ..	¼ cup	45	1	.14	1.6	15	A, B & C
Custard ‡ .....	⅔ cup	6 oz.	10	10	11.6	175	A, B & C
Toast or stale bread	1 slice	20	1.9	.18	10	50	A & B
Butter .....	1 pat §	13	.13	11	....	100	A
Milk .....	⅝ glass	5 oz.	4.7	5.7	7.2	100	A, B & C
TOTAL .....			20.43	27.17	52.4	540	
<i>Supper: 6 P. M.</i>							
Apple sauce .....	½ cup	120	.48	.6	18	75	B & C
Cream of wheat ....	½ cup	20	2	.16	14.7	65	A & B
Toast .....	1 slice	20	1.9	.18	10	50	A & B
Milk .....	⅝ glass	5 oz.	4.7	5.7	7.2	100	A, B & C
TOTAL .....			9.08	6.64	49.9	290	
TOTAL FOR DAY ....			46.31	45.43	150.6	1200	

\* Cream of wheat cooked.

† Toast, 1 slice: 4" × 4" × ½".

‡ Custard recipe 12½ ounces: { 8 ounces of milk ..... 160 calories  
 2 eggs ..... 150 calories  
 1 tablespoonful sugar ..... 50 calories

§ Butter, 1 pat: 1" × 1" × ¼", or 1 tablespoonful.

360 calories



## A DAY'S DIETARY FOR A CHILD OF FIVE YEARS

Approximate value of a day's diet { 15 per cent Protein, 56 grams, 225 calories  
 35 per cent Fat, 58 $\frac{1}{3}$  grams, 525 calories  
 50 per cent Carbohydrate, 187 $\frac{1}{2}$  grams, 750 calories

Fuel value: 1500 calories

Food	Amount	Weight, Grams	Pro- tein, Grams	Fat, Grams	Carbo- hydrate, Grams	Cal- ories	Vitamins
<i>Breakfast: 8 A. M.</i>							
Orange .....	I	.....	1.6	.27	22.79	100	B & C
Oatmeal .....	$\frac{1}{2}$ cup *	18 †	3	1.35	12	75	A & B
Egg .....	I	57	6.8	5.35	....	75	A
Bread (whole wheat) ..	I slice ‡	20	2	.18	10.18	50	A & B
Butter .....	$\frac{1}{2}$ tablespoon §	6.5	.06	5.5	....	50	A
Milk .....	I glass	6 oz.	5.7	6.9	8.6	120	A, B & C
TOTAL .....			19.16	19.55	53.57	470	
<i>Dinner: I P. M.</i>							
Cream of pea soup	$\frac{3}{8}$ cup	6 oz.	5.6	5	10	100	A, B & C
Scraped beef ¶	2 tablespoons	I oz.	5.5	2	....	40	C (?)
Baked potato .....	I medium	149	2.7	.15	22	100	B & C
Canned tomatoes .....	$\frac{1}{2}$ cup	110	1.3	.22	4.4	25	A, B & C
Bread (graham) ...	I slice	20	2	.18	10.18	50	A & B
Butter .....	I tablespoon	13	.13	11	....	100	A
Rice pudding **	$\frac{1}{2}$ cup	5 oz.	7	5.4	23	155	A, B & C
TOTAL .....			24.23	23.95	69.58	570	
<i>Supper: 6 P. M.</i>							
Apple sauce ††	$\frac{3}{4}$ cup	160	.64	.8	34	150	B & C
Bread (graham) ...	I slice	20	2	.18	10	50	A & B
Corn bread .....	I serving	40	3.04	1.18	17.8	100	B
Milk .....	$\frac{1}{2}$ glass	4 oz.	3.7	4.5	5.6	80	A, B & C
Butter .....	$\frac{1}{2}$ tablespoon	6.5	.06	5.5	....	100	A
TOTAL .....			9.44	12.16	67.4	480	
TOTAL FOR DAY ....			52.83	55.66	190.55	1520	

\*  $\frac{1}{2}$  cup cooked.

† Uncooked.

‡ Bread, I slice: 4"  $\times$  4"  $\times$   $\frac{1}{2}$ ".

§ Butter, I pat: 1"  $\times$  1"  $\times$   $\frac{1}{4}$ " or I tablespoonful.

¶ Scraped beef, steak or roast.

|| Pea soup recipe: 4 oz. milk, 2 oz. peas.

\*\* Recipe: 2 $\frac{1}{2}$  oz. milk,  $\frac{1}{2}$  egg, 2 teaspoonfuls sugar,  $\frac{1}{3}$  oz. rice (155 calories).

†† Recipe: 5 oz. apples, I tablespoonful sugar.

## ANOTHER CALCULATION ON SAME BASIS FOR CHILD OF FIVE YEARS

Protein, 55 grams; Fat, 55 grams; Carbohydrate, 190 grams

Fuel value: 1500 calories

Food	Amount	Weight, Grams	Pro- tein, Grams	Fat, Grams	Carbo- hydrate, Grams	Cal- ories	Vitamins
<i>Breakfast: 8 A. M.</i>							
Orange juice .....	½ cup	100	....	....	10.8	43	B & C
Cream of wheat ....	¾ cup	20	2.2	.3	16	75	A & B
		150 cooked					
Sugar .....	1 teaspoon	5	....	....	5	20	
Egg (poached) ....	1	60	6	6	.....	75	A
Bread (toast) ....	1 slice	20	1.86	.24	10.54	50	A & B
Butter .....	½ pat	5	.05	4.25	.....	38	A
Milk .....	6 oz.	150	4.95	6	7.5	104	A, B & C
TOTAL .....			15.06	16.79	49.84	405	
<i>Dinner: 1 P. M.</i>							
Chicken e.p.* .....	1 piece †	40	8.6	1	.....	60	
Potato e.p. ....	¾ cup	150	3.3	.15	28	125	B & C
Carrots .....	½ cup	100	1.1	.4	9.3	45	A, B & C
Canned string beans.	½ cup	100	1.1	.1	3.8	23	A, B & C
Bread .....	1 slice	20	1.86	.24	10.54	50	A & B
Butter .....	1 pat	10	.1	8.5	.....	77	A
Custard (baked or soft) .....	¾ cup	130	6.3	7	15	140	A, B & C
Milk .....	6 oz.	150	4.95	6	7.5	104	A, B & C
TOTAL .....			27.31	23.39	74.14	624	
<i>Supper: 6 P. M.</i>							
Cream of pea soup..	1 cup	205	7	4.27	19	140	A, B & C
Crackers ‡ .....	2	15	1.47	1.36	12	63	A & B
Bread (graham) ....	1 slice	20	1.78	.36	10.42	52	A & B
Butter .....	½ pat	5	.05	4.25	.....	38	A
Baked apple § ....	medium sized	130	.39	.39	19.04	80	A, B & C
Milk .....	6 oz.	150	4.95	6	7.5	104	A, B & C
TOTAL .....			15.64	16.63	67.96	477	
TOTAL FOR DAY ....			58.01	56.81	191.94	1506	

\* E.p. is the abbreviation for edible portion.

† 3" × 4" × ¼".

‡ Cracker 2½" × 2½".

§ Baked apple includes sugar.

## A DAY'S DIETARY FOR A CHILD OF SEVEN YEARS

Approximate value for day's diet { 15 per cent Protein, 63¾ grams, 255 calories  
 35 per cent Fat, 66 grams, 595 calories  
 50 per cent Carbohydrate, 212½ grams, 850 calories

Fuel value: 1700 calories

Food	Amount	Weight, Grams	Pro- tein, Grams	Fat, Grams	Carbo- hydrate, Grams	Cal- ories	Vitamins
<i>Breakfast: 8 A. M.</i>							
Prunes .....	2 large	33	.7	....	24	100	A & B
Cream of wheat ....	½ cup *	15 †	1.5	.12	10.5	50	A & B
Egg .....	1	57	6.8	5.35	....	75	A & B
Bacon .....	2 slices	15	3.3	9.64	....	100	
Toast (whole wheat)	1 slice ‡	20	2	.18	10.18	50	A & B
Butter .....	½ pat §	6.5	.06	5.5	....	50	A
Milk .....	1 glass	6 oz.	5.7	6.9	8.6	120	A, B & C
TOTAL .....			20.06	27.69	53.28	545	
<i>Dinner: 1 P. M.</i>							
Roast lamb .....	3 tablespoons	50	10.21	6.58	....	100	
Rice .....	½ cup	107	2	.06	23	100	
Peas .....	½ cup	100	6.99	.5	16.88	100	A, B & C
Carrots .....	½ cup	100	1.1	.4	9.3	45	A, B & C
Bread .....	2 slices	40	4	.36	20	100	A & B
Butter .....	1 tablespoon	13	.13	11	....	100	A
Canned peaches ....	2 medium	100	.7	.1	11.5	50	
Milk .....	1 glass	6 oz.	5.7	6.9	8.6	120	A, B & C
TOTAL .....			30.83	25.9	89.28	715	
<i>Supper: 6 P. M.</i>							
Apple sauce .....	¾ cup	160	.64	.8	35	150	B & C
Bread (graham) ...	2 slices	40	4	.36	20	100	A & B
Milk .....	1 glass	6 oz.	5.7	6.9	8.6	120	A, B & C
Butter .....	¾ pat	10	.1	8	....	75	A
TOTAL .....			10.44	16.06	63.6	445	
TOTAL FOR DAY ....			61.33	69.65	206.16	1705	

\* Cooked.

† Uncooked.

‡ Toast, 1 slice: 4" × 4" × ½".

§ Butter, 1 pat: 1" × 1" × ¼", or 1 tablespoonful.

## ALTERNATE DIETARY FOR A CHILD OF SEVEN YEARS

Protein, 61 grams; Fat, 70 grams; Carbohydrate, 207 grams

Fuel value: 1700 calories

Food	Amount	Weight, Grams	Pro- tein, Grams	Fat, Grams	Carbo- hydrate, Grams	Cal- ories	Vitamins
<i>Breakfast: 8 A. M.</i>							
Oatmeal .....	½ cup	15 dry 100 cooked	2.41	1.08	10.12	60	B
Sugar .....	1 teaspoon	5	....	....	5	20	
Apple sauce .....	½ cup	110	.4	.5	24.2	103	A, B & C
Bread* (for toast) ..	1 slice	20	1.86	.24	10.54	50	A & B
Butter † .....	½ pat	5	.05	4.25	.....	39	A
Egg (soft cooked) ..	1	60	6	6	.....	75	A & B
Milk (for cereal and to drink) .....	8 oz.	200	6.6	8	10	138	A, B & C
TOTAL .....			17.32	20.07	59.86	485	
<i>Dinner: 1 P. M.</i>							
Fish (as purchased) medium serving broiled .....	3" × 3" × 1"	100	8.4	.2		35	
Baked potato .....	1 medium	150	2.7	.15	22.05	100	B & C
Butter .....	1 pat	10	.1	8.5		77	A
Canned tomatoes ...	½ cup	100	1.2	.2	4	23	A, B & C
Bread .....	1 slice	20	1.86	.24	10.54	50	A, B & C
Butter .....	1 pat	10	.1	8.50		77	A
Rice pudding ‡ .....	¾ cup	120	4.89	5.09	36.2	217	A, B & C
Milk .....	6 oz.	150	4.95	6	7.5	104	A, B & C
TOTAL .....			24.2	28.88	80.29	683	
<i>Supper: 6 P. M.</i>							
Milk toast, milk and bread (toasted) ...	6 oz. 2 slices	150 40	4.95 3.72	6 .48	7.5 21.08	104 100	A, B & C
Prune whip § .....	1 cup	50	4.32	....	21.99	105	A, B & C
Bread .....	1 slice	20	1.86	.24	10.54	50	B & C
Butter .....	1 pat	10	.1	8.5	.....	77	A
Milk .....	6 oz.	150	4.95	6	7.5	104	A, B & C
TOTAL .....			19.90	21.22	68.61	540	
TOTAL FOR DAY .....			61.42	70.17	208.76	1708	

\* 1 slice bread: 3" × 2½" × ¼".

† 1 pat butter: 1" × 1" × ¼".

‡ Rice pudding includes rice, milk, sugar and egg.

§ Prune whip includes egg white, prunes.



## A DAY'S DIETARY FOR A CHILD OF NINE YEARS

Approximate value for day's diet { 15 per cent Protein, 75 grams, 300 calories  
 { 35 per cent Fat, 77 grams, 700 calories  
 { 50 per cent Carbohydrate, 250 grams, 1000 calories

Fuel value: 2000 calories

Food	Amount	Weight, Grams	Pro- tein, Grams	Fat, Grams	Carbo- hydrate, Grams	Cal- ories	Vitamins
<i>Breakfast: 8 A. M.</i>							
Banana .....	1	100	1.32	.61	22.3	100	B & C
Farina .....	¾ cup *	20 †	2.28	.3	15	75	A & B
Egg .....	1	57	6.67	5.22	....	75	A & B
Bacon .....	2 slices	15	3.31	9.64	....	100	
Toast .....	1 slice ‡	20	1.9	.18	10	50	A & B
Butter .....	1 pat §	13	.13	11	....	100	A
Milk .....	1 glass	6 oz.	5.7	6.9	8.6	120	A, B & C
TOTAL .....			21.31	33.85	55.9	620	
<i>Dinner: 1 P. M.</i>							
Chicken (broiler) .....		92	19.91	2.32	....	100	
Potato .....	1	150	2.7	.15	22	100	B & C
Lima beans (fresh) ½ cup		62	4.5	.42	13.5	75	A, B & C
Celery .....	2 stalks	50	.6	.05	1.7	10	A, B & C
Bread .....	2 slices	40	3.6	.46	20.39	100	A & B
Butter .....	1 pat	13	.13	11.1	....	100	A
Honey .....	2 tablespoons	30	.12	....	24.88	100	B
Blanc mange    .....	½ cup	.....	5	3.2	23	135	A, B & C
Milk .....	1 glass	6 oz.	5.7	6.9	8.6	120	A, B & C
TOTAL .....			42.26	24.60	114.07	840	
<i>Supper: 6 P. M.</i>							
Stewed apricots ¶ ..	½ cup	60	2.3	.5	40	175	
Bread .....	1 slice	20	2	.18	10	50	A & B
Corn bread .....	1 serving	40	3.04	1.18	17.8	100	B
Butter .....	1 pat	13	.13	11	....	100	A
Milk .....	1 glass	6 oz.	5.7	6.9	8.6	120	A, B & C
TOTAL .....			13.17	19.76	76.4	545	
TOTAL FOR DAY .....			76.74	78.25	246.37	2005	

\* Cooked.

† Uncooked.

‡ Bread, 1 slice: 4" × 4" × ½".

§ Butter, 1 pat: 1" × 1" × ¼", or 1 tablespoonful.

|| Blanc mange recipe: 1 cup milk, 1½ tablespoonful corn starch, 1½ tablespoonful sugar, white of egg.

¶ 1 teaspoonful of sugar used to sweeten.

## ALTERNATE DIETARY ON SAME CALCULATIONS FOR CHILD OF NINE YEARS

Protein, 76 grams; Fat, 78 grams; Carbohydrate, 248 grams

Fuel value: 2000 calories

Food	Amount	Weight, Grams	Pro- tein, Grams	Fat, Grams	Carbo- hydrate, Grams	Cal- ories	Vitamins
<i>Breakfast: 8 A. M.</i>							
Stewed prunes e.p...	6 medium size	50	.9	....	31.1	128	B
Farina .....	1 cup	30	3.3	.42	22.89	109	A & B
Sugar .....	1 teaspoon	5	....	....	5	20	
Bread (for toast)...	1 slice	20	1.86	.24	10.54	50	B & C
Butter .....	1 pat	10	.1	8.5	.....	77	A
Egg (soft cooked)...	1	60	6	6	.....	75	A
Milk (for cereal and to drink) .....	8 oz.	200	6.6	8	10	138	A, B & C
TOTAL .....			18.76	23.16	79.53	597	
<i>Dinner: 1 P. M.</i>							
Roast beef .....	1 slice *	60	12.78	4.74		93	A, B & C
Mash potatoes .....	¾ cup	150	3.3	.15	27.6	125	B & C
Fresh string beans...	½ cup	100	2.3	.3	7.4	42	A, B & C
Lettuce .....	2 leaves	30	.36	.09	.87	5	A, B & C
Fresh tomato (raw)	1 medium	100	.9	.4	3.9	23	A, B & C
Bread .....	2 slices	40	3.72	.48	20.84	104	B & C
Butter .....	1 pat	10	.1	8.5		77	A
Chocolate blanc mange † .....	¾ cup	130	5.46	6.89	27.77	195	A, B & C
Milk .....	6 oz.	150	4.95	6	7.5	104	A, B & C
TOTAL .....			33.87	27.55	95.88	768	
<i>Supper: 6 P. M.</i>							
Egg (poached) ....	1	60	6	6	.....	75	A
Spinach .....	½ cup	100	2.1	.3	3.2	24	A, B & C
Bread (toasted) ....	1 slice	20	1.86	.24	10.54	50	B & C
Butter .....	1 pat	10	.1	8.5	.....	77	A
Corn bread .....	1 piece ‡	80	6.08	2.36	35.6	200	A, B & C
Fresh strawberries .	½ cup	100	1	.6	7.4	39	C
Sugar .....	2 teaspoons	10	....	....	10	40	
Milk .....	8 oz.	200	6.6	8	10	138	A, B & C
TOTAL .....			23.74	26	76.74	643	
TOTAL FOR DAY .....			76.37	76.71	252.15	2008	

\* 1 slice: 4" × 4" × ½".

† Chocolate blanc mange includes milk, sugar, cocoa and cornstarch.

‡ 1 piece: 2" × 2" × 2".

## A DAY'S DIET FOR A CHILD OF TWELVE YEARS

Approximate value for day's diet { 15 per cent Protein, 94 grams, 375 calories  
 35 per cent Fat, 97 grams, 875 calories  
 50 per cent Carbohydrate, 312 grams, 1250 calories

Fuel value: 2500 calories

Food	Amount	Weight, Grams	Pro- tein, Grams	Fat, Grams	Carbo- hydrate, Grams	Cal- ories	Vitamins
<i>Breakfast: 8 A. M.</i>							
Raspberries .....	¾ cup	151	2.57	1.51	19.08	100	
Oatmeal .....	½ cup *	18 †	3	1.35	12	75	A & B
Egg .....	1	57	6.8	5.35	....	75	A
Bread (whole wheat) ..	2 slices ‡	40	4	.36	20	100	A & B
Butter .....	1 pat §	13	.12	11	....	100	A
Sugar .....	1 tablespoon	12	....	....	12	50	
Cream (20 per cent) ½ cup		50	1.28	9.5	2.3	100	A & B
TOTAL .....			17.77	29.07	65.38	600	
<i>Dinner: 1 P. M.</i>							
Roast beef (lean) ..		100	20	7.6	....	150	
Baked potato .....	2 medium	300	5.4	.3	44	200	B & C
Beets .....	½ cup	100	1.7	.11	10.5	50	B
Bread (whole wheat) ..	2 slices	40	3.9	.36	20.2	100	A & B
Butter .....	1½ pats	20	.2	15.5	....	150	A
Canned pineapple .....		100	.39	.67	36.5	150	
Milk .....	1 glass	6 oz.	5.7	6.9	8.6	120	A, B & C
TOTAL .....			37.29	31.44	119.8	920	
<i>Supper: 6 P. M.</i>							
Creamed chipped beef ¶ .....	¾ cup	.....	29.5	15.5	11.4	300	A, B & C
Bread (whole wheat) ..	3 slices	60	5.8	.54	30.3	150	A & B
Butter .....	1 pat	13	.13	11	....	100	A
Jelly .....	2 tablespoons	40	.45	....	32.4	135	
Baked apple .....	1 medium	160	.64	.8	23	100	B & C
Sugar .....	1½ teaspoons	20	....	....	20	75	
Milk .....	1 glass	6 oz.	5.7	6.9	8.6	120	A, B & C
TOTAL .....			42.22	34.74	125.7	980	
TOTAL FOR DAY .....			97.28	95.25	310.88	2500	

\* Cooked.

† Uncooked.

‡ Bread, 1 slice: 4" × 4" × ½".

§ Butter, 1 pat: 1" × 1" × ¼", or 1 tablespoonful.

|| Table cream or 20 per cent.

¶ ½ cup chipped beef + ½ cup cream sauce (1 cup milk, 1 tablespoonful flour, 1 tablespoonful butter).

## CHAPTER IX

### UNDERDEVELOPMENT AND OVERDEVELOPMENT

We have seen in the discussion of the relation of height to age, that in measuring large numbers of children in order to determine an average, there is considerable individual variation on both sides of the plotted curve which represents the average. This variation, as a rule, amounts to only a few inches, but in some of the studies children have been found in each series who differ from the average to a considerable extent; a number being as much as seven inches above or below the average. It has also been shown that racial and familial influences account for some of the variations. Other factors probably play a part in determining the height of the individual. How many of the short children subsequently reach the average height for their age, it is not possible to state, since no studies are available where such children have been followed to maturity. That many of these short children belong in the underdeveloped class there can be little doubt; and that a number of them remain short throughout life is fairly certain, judging entirely, however, from observation. In addition to this there are a large number of individuals, both children and adults, who are so short as to be noticeable among their associates. All individuals of unusually low stature are to be regarded as in the class of the underdeveloped.

Underdevelopment, infantilism, and dwarfism are some of the terms commonly used to designate marked deviation below the average, or so-called normal standard of height. These terms are not synonymous, but are often used loosely to describe a number of conditions characterized by low stature. There are certain more or less definite differences—morphologic and anthropologic—between the various types of dwarfism; and some morphologic and clinical differences between several types of infantilism. On the other hand there are many features of resemblance between all of the types, so that it is often difficult to differentiate them sharply. Some cases of underdevelopment are distinctly congenital and in some instances due to heritable traits, while others apparently are the result of environmental influences, such as intra-uterine disease, or other conditions acting during antenatal life; and postnatal disease or malnutrition.

**Dwarfism.**—The failure of an individual to develop in size from a child to an adult, morphologically, is best illustrated by the dwarf. Dwarfism is an interesting and relatively rare condition, in which there is a general underdevelopment. There are several varieties of dwarfs described, which differ materially from the “true dwarf” or “atelirosis.” The true dwarf is small in all dimensions, and all the organs are correspondingly and uniformly small. Other types of dwarf are not so uniform in dimension (achondroplasiac), and are often distorted (rachitic), or have an edematous countenance (myxedematous). There are a number of distinguishing characteristics of these individuals, only a few of which will be mentioned. They range in height from 2 to 4 feet, and are usually described as miniature adults. This is true so far as the symmetrical proportions of the body are concerned, but they retain the relative proportions of the child. The head is relatively large, the cranium being out of proportion to the rest of the skull, and the infantile facies retained. The neck is short. The males grow little or no facial hair, and the females rarely develop decided feminine features, retaining the asexual ones of the child. The relation of the extremities to the torso remains infantile, though both are of normal shape. The feet and hands are noticeably small. Secondary sex characteristics seldom develop and sterility is the rule, though exceptions are not rare. The mentality of dwarfs varies within wide limits. Most of them are slightly retarded, some are distinctly feeble-minded, while some have been of exceptional mental capacity.

Some observers have classified dwarfism chronologically, that is according to the age at which development was interrupted; this is probably not justifiable, however, since this fact appears to have no influence on the appearance or the characteristics. Some dwarfs are born small, while others are normal at birth, interrupted development not occurring until later; during the first year, the second or occasionally not until the fifth year. Some dwarfs grow normally for the first few years of life and then grow slowly for a number of years, while a few have been known to begin growing suddenly after a prolonged period of complete inhibition. Most dwarfs die young, usually in infancy, others live the allotted span of life; while a few have lived to remarkably advanced ages.

*Etiology.*—Our knowledge of the cause of true dwarfism is entirely speculative as yet. That a lack of “growth impulse” exists is almost axiomatic. Why, no one knows. Apparently all of the body cells are affected, and yet from autopsy findings it is evident that all



are not affected equally, since "in the same dwarfs mature organs and functions may be found, with others which are puerile, some which are infantile, and still others which are fetal." The time of union of the



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FIG. 33.—UNDERDEVELOPMENT DUE TO CHONDRODYSTROPHY (ACHONDROPLASIA).

The large head often seen in these cases is well shown here. There is a decided suspicion of hydrocephalus in this instance, but is probably not the case, since the forehead is not bulging enough and the upper part of the whites of the eyes are not conspicuous.



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FIG. 34.—ACHONDROPLASTIC DWARF.

(From Chapin and Royster, *Diseases of Children*.)

epiphysis often determines stature; premature union causing arrested growth, and delayed union causing overgrowth. Embryos have been found which are very much smaller than the average of their age, and others which are far larger than the average; such deviation may result in dwarfism or giantism (Jordan). It is possible that dwarfism may

result from dysfunction of those organs of internal secretion which are supposed to influence stature, but at present there is no proof.

In addition to true dwarfism there are several conditions resulting in underdevelopment with dwarflike stature. The most striking of these, and the one most frequently mistaken for true dwarfism is achondroplasia. The most marked feature of this type of underdevelopment

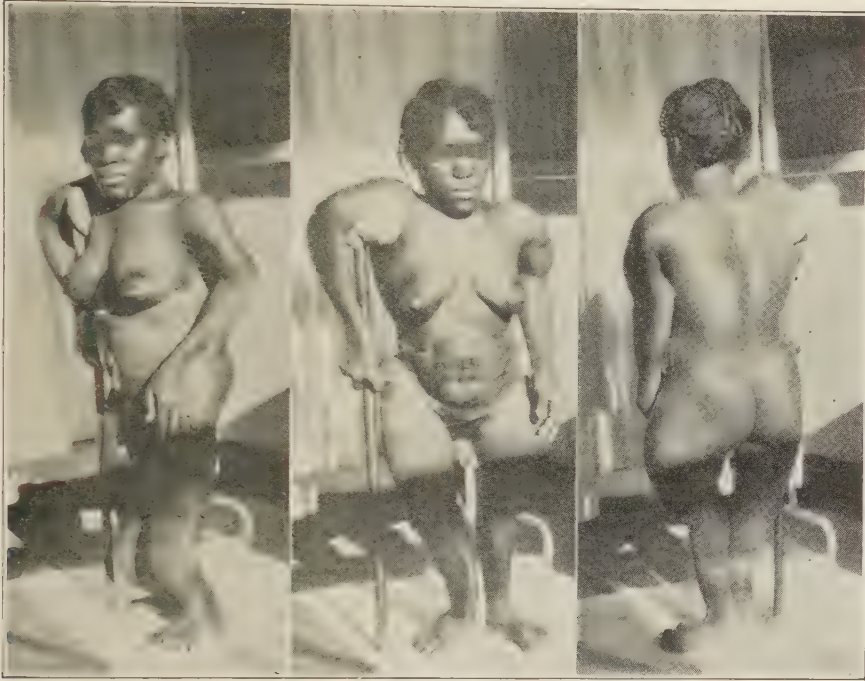


FIG. 35.—THREE VIEWS OF A RACHITIC DWARF, TWENTY-THREE YEARS OLD AND FORTY-SEVEN INCHES HIGH.

She has just recovered from a cesarian section.

is the shortness of the extremities. The torso participates in the process but not to the same extent as the extremities. The effect is that the extremities appear abnormally short in comparison with the trunk, presenting a rather grotesque appearance. The head in extreme cases appears large, and out of proportion to the rest of the body, and in fact there is sometimes an accompanying moderate hydrocephalus. The deformities are "due to an abnormal process of endochondral ossification at the junction of the epiphysis and diaphysis. The principal change is a defective formation of rows of cartilage cells in the columnar zone." Sometimes an overgrowth of periosteum occurs, which wedges its way between the epiphysis and the diaphysis from

the periphery toward the axis of the bone. This condition is essentially congenital, and most cases are apparent at the time of birth. In many instances the parents of achondroplasiacs are undersized and may be affected with the same condition, though not to such an extent as to be



FIG. 36.—TYPICAL CRETIN, CLASSED WITH DWARFISM.

(From Chapin and Royster, *Diseases of Children*.)

definitely demonstrable. The mental state varies, in most instances it is unimpaired, in fact many of these individuals are quite bright, in others however there is a definite amount of mental retardation. It would appear that those having an accompanying hydrocephalus show mental retardation. The facial expression in marked cases is quite characteristic, being "heavy," expressionless, or apathetic. The nose is somewhat bulbous, with flattening at the bridge. The teeth are late in erupting and the fontanels in closing. A marked lumbar lordosis is usually present, due to abnormal thickening of the anterior part of the bodies of the lumbar vertebræ. The frequent confusion of this condition with rickets is due to the short and thick bones with enlarged epiphysis. There are other characteristics, not specific for this condition, but observed in a number

of other conditions of underdevelopment of body or mind, notably the trident hand.

Extreme deformity from rickets often results in dwarfism. This is in no sense a true dwarfism, but results from the interruption to bony growth, and that of the body as a whole, described in the section on rickets. Distortion of the bones further accentuates the diminished stature.

Myxedematous dwarfs or cretins are interesting, but are merely mentioned, being discussed in another volume of the series.

**Infantilism.**—Infantilism is a term applied to a large group of children who are distinctly undersized. There is a retardation of bodily development out of proportion to chronological age. The stature is small and underweight for the height is the rule. The childlike falsetto voice is retained, and poor or retarded sexual development the rule. The mentality is usually unimpaired, in fact many of these individuals are exceedingly bright. Some of this mental brightness is more apparent than real, since the chronological age is often overlooked, and such children appear precocious for their diminutive size.

A number of types of infantilism have been described, differing in one or more particulars, but it is doubtful if very sharp distinction can be made. The usual descriptions of underdevelopment, of all types and kinds in which the term infantilism is used, leave much to be desired and cause confusion in the mind of the reader. Underdevelopment of the kind usually described as infantilism may be regarded as symptomatic rather than structural or essential. These cases are probably always secondary to some disease or derangement of metabolism and there is absence of any sugges-

sion of heredity. Infantilism should not be employed to designate arrest of growth alone, but in order that the term be applied aptly there should also remain characteristics of an earlier age period. Although all classifications at present are open to serious objection, there are certain characteristics on which these classifications have been based, which differ in many respects from each other.

In the group classed as the Lorain type are those who are small and resemble a younger individual; on closer examination, however,



FIG. 37.—CHILD FOUR YEARS AND SIX MONTHS OLD, THIRTY-FIVE AND ONE-HALF INCHES TALL, WEIGHING TWENTY-THREE POUNDS.

This child is to be classed as a case of infantilism. There is nothing in the history to account for the condition. The child was small at birth and has remained so ever since. There has never been any digestive disturbance, and the food has apparently been adequate.



the proportions are more like those of the adult; particularly in respect to the relation of extremities to torso. The head is small and in proportion to the rest of the body. The secondary sex characteristics, however, are either wanting or appear late, and then to a very slight degree. The mental development is either normal or very slightly retarded. These individuals are children in size, but adults in every other respect except that of sex development. They are rarely robust, the vital processes being decidedly below par. They are affected with frequent intercurrent infections. In general these individuals approach dwarfism more closely than they do infantilism. One of the most striking points of differentiation is that they are never congenital, development being arrested after an apparently normal period of growth.

The etiology of this type of infantilism is not known with any degree of definiteness. Among the conditions which have been thought to cause it, whether directly or through interference with nutrition, or in some manner causing a retardation of the growth impulse are: inherited syphilis, lymphatism; such infections as malaria and tuberculosis, and any condition which interferes materially with nutrition.

Myxedematous infantilism is a type which is characterized by shortness of stature, moderate obesity, undeveloped sexual organs, and infantile proportions of the body. The extremities are short, thick and round, and the torso is noticeably large. This is known as the Brissaud type, and is undoubtedly due to thyroid deficiency. It differs from cretinism in several respects. None of the characteristics are so well marked as in cretinism. Mental impairment, though generally present, is less marked. The most striking difference between the two conditions, however, is that while cretinism is manifest early in the first year of life, myxedematous infantilism is seldom apparent until the third year and often later than this. That this condition is a true hypothyroid deficiency is attested by the fact that improvement promptly follows thyroid administration. As in cretinism, however, it must be kept up, and interruption of treatment is followed by a return of all symptoms. Complete recovery must not be expected.

The type of infantilism of the greatest interest is what is called intestinal infantilism. This condition follows prolonged states of malnutrition of any kind, but the condition with which it is most often associated is chronic intestinal indigestion or so-called celiac disease, in fact this association is so constant that arrested growth is expected as a symptom of celiac disease. These patients suffer from repeated



digestive disturbances particularly referable to the intestinal tract. There is an alternation between diarrhea and constipation. There is a marked fat intolerance, and during the exacerbations of diarrhea the



FIG. 38.—INFANTILISM.

Boy seven years old, height thirty-three and one-half inches (fourteen and one-half inches below normal for age), weight twenty-five and one-half pounds. Proportions all good. Mentality apparently good, but he was so small for his age that he appeared brighter than he really was. He was an epileptic. There was nothing other than what is recorded to account for his size. This case was reported in the *Virginia Medical Monthly* for September, 1925, as a case of situs transversus.

stools show evidence of fat indigestion, such as the presence of free fat, fatty acids, and soaps. There is accumulation of gas in the intestinal tract, which largely on account of the lowered muscular power of

the intestines causes great distention of the abdomen, a prominent feature of the condition. In spite of the lowered muscle power there is frequent expulsion of flatus, which is foul smelling, as are the stools themselves. The character of the stools varies. At times they are of a thin soupy consistency, while at others they are mushy or even sometimes scybalous. They are usually of very light clay color. There is in addition to the arrest of growth a marked emaciation, the extremities showing this to a pronounced degree; this with the distended abdomen gives the appearance of a gnome, which in some cases is decidedly grotesque. Anemia may be extreme; the musculature is flabby, with a decided lack of endurance, the sufferers becoming fatigued on the slightest exertion. In sharp contrast with most other forms of arrested growth the mentality is not only not retarded but the patient is usually precocious. These cases have been studied quite extensively, particularly by Herter who found an absence of the normal intestinal flora, and a Gram-positive organism in excess. It is likely that regardless of the exact etiology of the condition, the absorption of toxins formed in the intestines as a result of certain changes, fermental or otherwise, plays a part in its development. This form, to which has been given a name (Herter's infantilism), represents a type of process found in a number of digestive derangements; in fact it is entirely likely that any prolonged state of malnutrition resulting from extreme disorders of digestion and metabolism is capable of producing an arrest of growth and development. Closely allied if not identical is the so-called pancreatic type of infantilism. The theory of the influence of the pancreas was advanced after the treatment of a patient with arrested development, particularly of the generative organs, with remarkable benefit with pancreatic administration.

Several other forms or types of infantilism have been described, but are probably not sufficiently sharply defined to justify a separate classification. Cardiac infantilism, which is an arrest of growth and development due to congenital or acquired heart disease, has been placed in a separate classification. Such arrest occurs and merits attention, but hardly justifies classification as a type. It belongs in that group caused by diseases or conditions interfering with nutrition to an extent sufficient to arrest growth and development.

A type of infantilism known as pituitary infantilism, or Fröhlich's syndrome, occupies a peculiar position, in that there is a marked increase in adiposity, accompanied with a perpetuation of certain infantile characteristics. This condition forms a link between underdevelop-

ment and overdevelopment. It is in most instances associated with tumor of the pituitary, though there may be only dysfunction of that gland. It may appear quite early in life or develop at any time later. When there is definite tumor of the pituitary, headache is constant or intermittent; headache is not the rule with dysfunction. There are two outstanding physical characteristics: marked obesity, and a lack of sexual development. The obesity may reach a stage where the over-



FIG. 39.-FRÖHLICH'S SYNDROME.  
(From Chapin and Royster, *Diseases of Children*.)

weight is so great that it is difficult for the individual to handle himself with any degree of ease or comfort. This obesity is general, but in certain localities fat is deposited in extraordinary amounts; the mammary region is fat and pendulous, the pelvic girdle has excessive deposits of fat, the buttocks and thighs are enormously large and the mons veneris is often so pendulous that the genital organs are scarcely visible.

The sexual organs are infantile in appearance and in some instances diminutive. There is no hair in the pubic region, and none in the axilla. If hair grows on the face at all it is scant. Polyuria is a frequent occurrence, being analogous to diabetes insipidus. The carbohydrate and glucose tolerance are vastly increased. As a rule the stature

is below the average, but in some cases it is above. The sufferers are as a rule sluggish both physically and mentally; marked asthenia is generally present and a large amount of sleep is required.

As stated already, this condition is undoubtedly caused by some derangement of the pituitary, though the symptoms and signs are in some cases contradictory. The overgrowth of the body, when accompanying a sexual infantilism, might cast doubt on this theory, but Cushing accounts for this phenomenon as a possible activation of the anterior lobe of the pituitary, with insufficiency of the posterior lobe. No treatment apparently accomplishes anything in the way of improvement.

In sharp contrast to the various conditions just discussed, in which an arrest of growth is a prominent feature, we have a rather small and unimportant group of individuals who are very much above the average height. Just how much height in excess of the average should be considered as constituting an abnormality, or should be placed in the class of giantism, is difficult to estimate. It has been shown that many individual children are below or above the average height for age, and variations of as much as seven inches are not rare. In the adult also it is not unusual to see individuals who are many inches taller than the average of the race. There appears to be a factor for tallness in certain families; and in more or less circumscribed areas of the country tall men are the rule. Giantism is a term applied to adult individuals who are "abnormally" or "excessively" tall. In childhood the term may be applied with some propriety to those who are noticeably beyond the average height for their age, or to those who grow at an excessive rate. In childhood excessive height is usually accompanied by some degree of overweight, although there are some who are inordinately thin for an abnormal height, and who are said to have outgrown their weight and strength. In some instances children are born much taller and much heavier than the average. When this excess in either particular is not very great, it is generally supposed that the infant has remained in the uterus beyond the normal period of gestation. Where the overheight and overweight approach dimensions bordering on monstrosity, it is likely that other factors enter into the etiology of the condition. Some children who are unusually large at birth continue to grow and develop well in advance of their chronological age, and reach adult life noticeably large individuals. In other cases infants are born average individuals in every respect and at some time begin to grow at an accelerated rate and reach puberty or adult life well above the average. In oversized individuals there are usually other



been alluded to that the time of union between the epiphysis with the diaphysis determines stature; in some cases, therefore, it is only natural to presume that a delay in this union accounts for some cases of giantism. There is a marked familial tendency, in many instances; on the other hand some of the tallest giants were born of normal parents.



FIG. 42.—NEGRO GIRL JUST APPROACHING PUBERTY.

Tremendously overweight. Voracious appetite. Special fondness for sweets. Local deposition of fat in addition to general obesity. Good-natured. Low grade mentality. Probably endocrine.

Whether belonging to the class of the overdeveloped or to the over-nourished, or to endocrine dyscrasias, obesity is one of the most interesting conditions with which we have to deal, and at the same time one of the most difficult to handle, when it is advisable to reduce the amount of fat. Obesity is described as an excessive deposition of fat in the subcutaneous region and in various organs of the body. Just what may be regarded as "an excessive amount of fat" is a matter of much debate.



Emerson and others have been accustomed to regard 20 per cent of weight in excess of the average for height as constituting obesity.



FIG. 43.—GIRL THIRTEEN AND ONE-HALF YEARS OLD, SIXTY-ONE INCHES TALL, WEIGHING ONE HUNDRED AND NINE POUNDS.

This weight is just one pound above average for height. This girl had been very much stouter, and had been dieted to some extent, and had had regulated exercises. The fat deposits had been very much more marked than at the time of the picture. She was bright, alert and generally accomplished. The diet and exercise reduced her weight some, but had no effect on the fat deposits. Just as soon as puberty was established her whole contour changed; she became becomingly slender and the unusual fat deposits disappeared. At the first appearance of menstruation all dieting and prescribed exercises were discontinued.

There are some children who look fat while weighing less than this and others who do not appear fat when weighing more than 20 per cent over the average.

The opinion of an experienced observer of children is worth more than a fixed figure; at the same time most children who weigh 20 per cent or more in excess of the average for height are in fact obese and appear so. Some infants are born weighing far in excess of the normal at birth, and persist in remaining in excess throughout the balance of life. In some of these cases the individuals are also taller than the average. This has already been discussed in this section. The type of obesity to which we refer at this time is rather apt to develop somewhat later—from the fourth or fifth year to puberty. There are two types of obesity: those who are uniformly and evenly overfat with no other abnormality presenting; and those in whom in addition to a general excessive amount of fat, there are additional deposits in various localities, such as the mammary region, pelvic girdle, and mons veneris. This latter group are supposed to result from certain endocrine disturbances, and their similarity to certain other types of known endocrine origin (Fröhlich), so far as contour is concerned, makes this quite probable.

It is more than likely that a disproportion between the intake of food and the needs of the body exists in all of those of the former group and probably also in those of the latter group. The table on page 125 gives the caloric needs of the growing child. This takes into consideration the needs of the average child of normal development and activity. In the vast majority of obese children careful checking of the daily food will elicit the fact that the food intake is much in excess of this requirement. In some cases the total amount of food may be slightly in excess of the requirements, but it will be found that the diet is not balanced; there is in such instances usually a deficiency of vegetables and fruits and an excess of fats and carbohydrates, such as bread, butter, pastry and candy. The habits of the individual must also be enquired into before a definite opinion can be given. The amount of food may be within normal limits or slightly in excess but the child may lead a very inactive life. Thus the food may fail to meet the body requirements, and consequently be unbalanced in one of several ways.

Does heredity play a part in the production of obesity? This is a question which has been much debated, and is a cause often assigned by the parents of a fat child. There can be no doubt that the parents of overweight children are often affected in the same manner. The conformation of the child may also be "exactly like that of the parent"; and yet we have no definite way at this time of proving or disproving

this point. It is perhaps true that familial tendencies to obesity do exist; or at least that heritable factors are present in some individuals which either promote a tolerance for carbohydrates and fats, or prevent

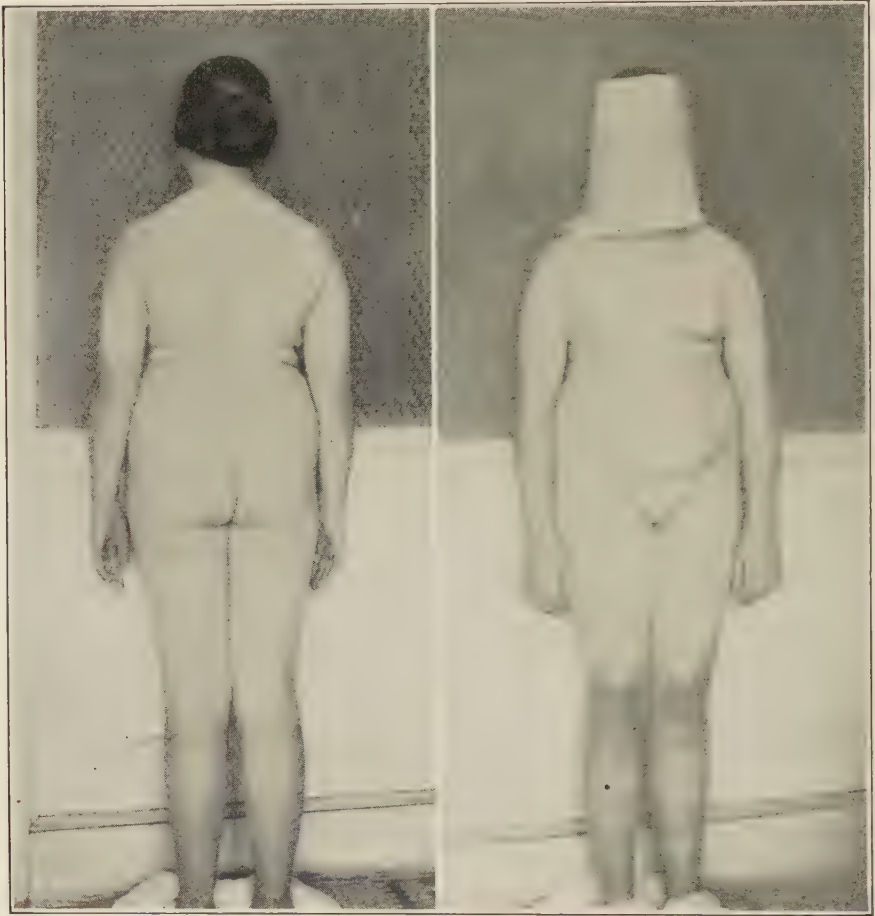


FIG. 44.—GIRL TWELVE YEARS AND FOUR MONTHS OLD, SIXTY-TWO INCHES TALL, WEIGHING ONE HUNDRED AND THIRTY-FIVE POUNDS.

Besides being generally obese, she has localized fat deposits. Her disposition was fractious and discontented. Diet and exercise improved her somewhat but entire lack of coöperation prevented much improvement. With the establishment of menstruation she became quite slender and well formed, the fat pads disappeared almost entirely and her contour became good. In fact she lost all appearance of marked obesity. Her disposition improved and she is well liked and does well in school work without urging.

adequate oxidation. In many instances, however, it is probable that the habits of overeating on the part of those of the older generation, are instilled into the offspring, through example, and through availability

at all times of an overabundance of fattening food. At present there is no reliable method of diagnosing obesity as of endocrine origin. Even the basal metabolism test has proved to be unreliable. The con-

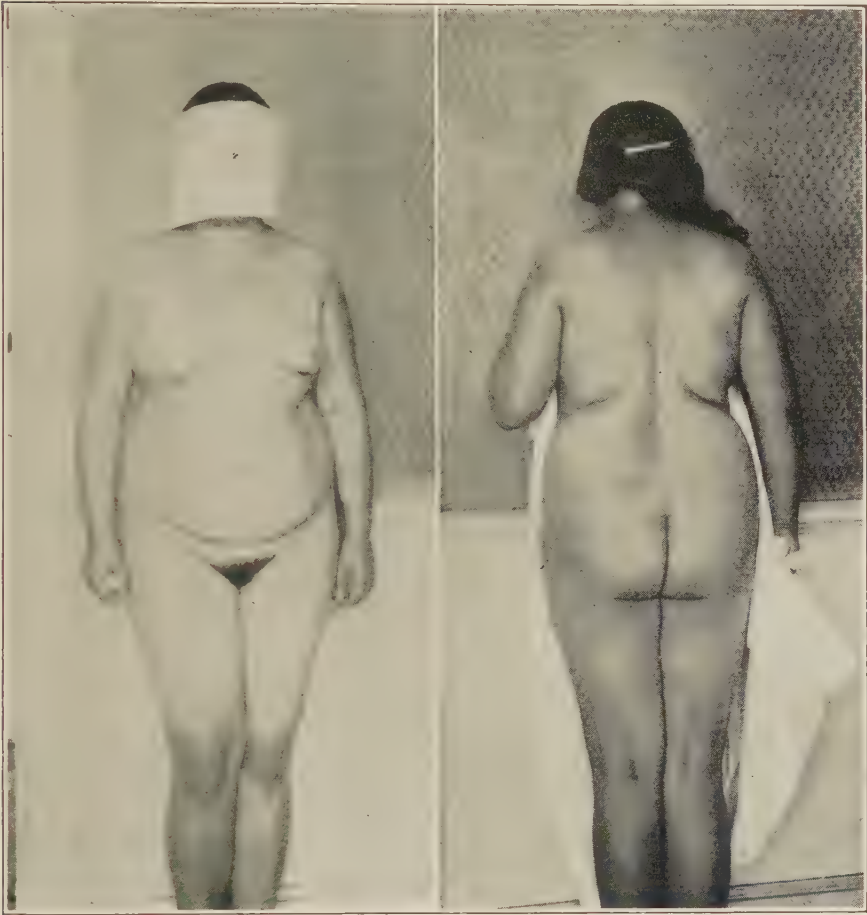


FIG. 45.—GIRL FIFTEEN YEARS OLD, SIXTY-FOUR AND THREE-FOURTHS INCHES TALL, WEIGHING ONE HUNDRED AND SIXTY-NINE POUNDS.

Normal weight for height one hundred and twenty-four pounds. This girl had large localized fat deposits in addition to general obesity. She was mentally sluggish, and of a rather surly disposition, rather the exception in this type. Menstruation had been established. Moderate dieting and regulated exercises were attempted, but since no coöperation was secured it was not determined whether anything could be accomplished. She was lethargic, never exerting herself in any way that could be avoided. She remained obese and unattractive.

tour and body measurements in certain cases suggest an endocrine disturbance. The irregular deposition of fat, a disproportion between the various body measurements, and a certain type of disposition strongly



suggest an endocrine origin. Endocrines are apt to be of a "sweet disposition," childish in their likes and dislikes, easily pleased or displeased, and generally possess a somewhat immature mind. They are usually described as having a "sweet face," which means in these cases a round fat face with the expression of a doll. In the majority of obese children, however, no definite diagnosis between types is possible, there being no sharp line of demarcation.

We have already noted that a fatigue posture is common with undernourished children. A faulty posture is often observed in obese children also. In some instances the fault is as great as in very thin individuals. Just why this is cannot be stated positively. All obese individuals have large abdomens which protrude to a considerable extent. This additional weight at that point causes the individual to lean backward and thus accentuates the lumbar curve. It must be further noted that irregular deposits of fat at certain points may give the impression of faulty posture which does not actually exist.

Obese children are sluggish, because of overweight and awkwardness, and consequently they take little or no exercise, and practically never engage in competitive sports. Their awkwardness is due not only to the excessive weight, but also the fact that most of them are knock-kneed and have pronated feet. They are usually sensitive and are often the butt for ridicule.

Although obesity often occurs during infancy and early childhood the most noticeable cases are those which occur just before or at puberty. In observing a large number of boys and girls at this age and especially those who were either markedly underweight or obese, I have observed many with and many without those characteristics which suggest endocrine disturbance. Girls are more frequently obese than boys, particularly near puberty. I have seen some whom I attempted to reduce within what were considered justifiable limits, and who responded and became more or less normal figures. Others who would not submit to treatment, or coöperated poorly became slender in spite of continuing to eat what they wanted. As a result of my own observations I am convinced that a prepuberal obesity often occurs which corrects itself without interference when sexual maturity is established. This suggests at least that certain endocrine influences act on the metabolism in one way during the years just before puberty, and in a directly opposite manner when this is established.

**Treatment.**—The first question which naturally arises is, Should the overweight child be deliberately reduced? This question is pertinent



and important. There are two main considerations in a discussion on this phase of the subject: the prevention of obesity, and its correction when once acquired. If prevention is considered seriously, and there can be little doubt that this is advisable, it should be begun with careful supervision of the food and food habits of the infant. The weight of the nursing infant should be watched carefully, and if it begins to increase at a rate much in excess of the normal or average curve, the amount of food should be reduced until the desired weight is reached. This can be done easily by reducing the length of each nursing. When observations can be made by weighing, before and after each nursing, the amount of food can be adjusted with accuracy to the needs of the infant. In the case of artificially fed infants this can be done with even greater ease, by following the directions in the chapter on the feeding of the normal infant, in order to feed by giving an adequate amount of protein and a sufficient number of calories. In older children directions given in a previous section on how to feed the normal child may be followed, the requirements met and the exact amount of food needed by the individual child given.

When we have regulated the amount of food taken by the child the only other consideration of importance is the regulation of the exercise, or the prescribing of sufficient amount to insure proper oxidation.

When it is deemed advisable to reduce the weight of an older child to any appreciable extent the same principles apply as in the prevention: namely, a reduction in total calories, and regulation of exercise. It is exceedingly inadvisable to attempt a rapid reduction, since the reaction is apt to be too great. These overfat children already have poor stamina and the vital forces are often low. The musculature is flabby and endurance is slight. It is exceedingly important that in reducing the total quantity of food, the protein needs of the body be kept up, and consequently the protein of the food should not be reduced. Since carbohydrates of all kinds are largely responsible for the excess fat, they should be reduced to a minimum, the fats also should be reduced but need not be to the same proportionate extent as the carbohydrates. A diet should be worked out along these lines and written down for the guidance of both parent and child. Such a diet can easily be devised by reference to the tables of food values on page 192. The retention of water probably has its effect on the weight, and as a rule fat individuals are excessive water drinkers. Of course the water intake may be reduced somewhat, but it is unwise to restrict its consumption too much; when the weight is reduced and the total amount of fat is very

much less than at the time of beginning the treatment, the desire for water is less.

No strenuous exercise should be advised at first. Walking is the best form of exercise for these people, but when taken alone is usually monotonous and it is difficult to induce the individual to persist in it. Setting up exercises carefully supervised and gradually increased are very beneficial; this is especially true when the class system is employed. Tennis is good at a later time when the muscles are harder, and golf and rowing are both excellent.

To what extent the endocrine system is responsible we do not know at this time. At all events it is exceedingly unwise to administer extracts in any form, unless carefully checked by estimation of the basal metabolism rate.

## CHAPTER X

### INANITION AND MALNUTRITION

Malnutrition is the state or condition of being improperly or insufficiently nourished. Included in this term are inanition, starvation, and obesity. Undernourished individuals are often called malnourished; but restriction of the use of the word to this class of individuals is an error. Since water is an essential element of the human body and is classed as food, a state of water malnutrition or inanition or dehydration often exists. Certain mineral salts are also essential to a state of well-balanced nutrition, and any disturbance of mineral metabolism must likewise be classed as malnutrition. Vitamins, or accessory food factors, promote in a manner not as yet fully understood, metabolic processes which when impaired by a deficiency of these factors produce symptoms definitely in the class of malnutrition. Thus we have included in the term malnutrition, undernutrition (inanition, marasmus, starvation); overnutrition (obesity, and certain dyscrasias having overdeposition of fat as a prominent manifestation); mineral-salt deficiency (rickets and other diseases); and the conditions due to avitaminosis (scurvy, pellagra, beriberi, xerophthalmia, etc.). In some of these conditions there is probably a combination of influences, for example, in the case of rickets, a faulty calcium and phosphorus metabolism due to a lack of vitamin D, or of sunshine, or of both. Sunlight therefore must be considered as a food factor, and certain conditions brought about, in part at least, by a deficiency of sunlight must be placed in the malnutrition class.

Malnutrition is best considered, clinically, in two divisions: (1) as it occurs in infancy, that is during the first two years of life, and (2) as it occurs in older children. In infancy undernutrition is manifested in three stages, acute inanition usually violent in its onset; a second condition not so acute nor severe usually called malnutrition, but to which a number of terms have been applied; and marasmus, a severe, extreme state of chronic undernutrition. On the other extreme are the overfat and often overdeveloped infants. In older children a state of malnutrition may show in any of the ways described for infants, but as a rule undernutrition is most usual and is what is meant when we

speak of the malnourished child. Here too, as infants, and with few exceptions, of greater significance we have the overnourished or obese child. The degree of malnutrition, with its accompanying nomenclature, depends on the severity or duration or both of the disarranged metabolic process.

**Acute Inanition.**—Acute inanition is loss of weight occurring with such rapidity that an acute condition which endangers life, may be reached in a very short time.

*Etiology.*—This condition occurs most frequently, in fact almost entirely, in young infants. Lack of food ingestion is by far the most usual cause. In very young infants, that is during the first two weeks or the period generally called newborn, some infants refuse to take any nourishment at all. They seem to be unable to learn to nurse. This occurs even where the infant is apparently well developed and quite strong. Premature infants are often too weak to nurse vigorously or even at all; while a number actually born at full term are physiologically premature and suffer in the same manner. Atelectasis and intracranial hemorrhage are also frequent causes of failure to nurse. Certain diseases are the cause of acute inanition either because of extreme weakness or an inability to properly assimilate ingested food. This is often the case with newborn congenital syphilitics. In older infants the condition may be caused by prolonged vomiting, such as from congenital pyloric stenosis, and this may occur at any time during the course of the stenosis from the beginning, during the first weeks of life, to the time when the stenosis subsides spontaneously or is operated on. Prolonged diseases, during which an insufficient amount of food is taken, cause a steady loss of weight, but during the course of such diseases there may develop at any time an acute inanition. This is apt to be the case where there is an insufficient retention of food, even though a sufficient amount is taken, as often occurs in severe and prolonged diarrhea. Congenital defects of structure which interfere with nursing or swallowing, or the free passage of ingested food, such as stricture, harelip or cleft-palate, are important factors in the development of acute inanition. Prolonged cases of indigestion, even though there has been slight loss of weight, may suddenly develop an acute inanition. Faulty absorption of food may act in the same manner. Some infants and children are possessed of inherent tendencies or peculiarities of digestive power, called intolerance or idiosyncrasy, which interfere with proper absorption and digestion of food and cause marked disturbance of nutrition. In some cases there appears to be

a lack of metabolic power on the part of the cells themselves; under these circumstances a poor state of nutrition develops and may become acute at any time. Certain toxic influences act in a similar manner. Thus it may be seen that the causes of this condition are many, and may act at any time during infancy. Typical inanition of the acute and



FIG. 46.—CASE OF ACUTE INANITION.

Infant aged seven months, height fifty-eight centimeters, weighing nine pounds two ounces.

severe variety, however, occurs most frequently during the early days or weeks of life.

*Symptoms.*—From whatever of the foregoing causes, the most prominent and serious symptom is the very rapid loss of weight. In some instances, especially due to vomiting, where the gravity of the case is not recognized promptly and proper treatment instituted, death occurs in a very few days; in such cases the loss of weight may amount to 50 per cent. In other cases there has been a gradual loss of weight, which has not been considered serious, and suddenly a state of acute inanition develops. "The pulse is weak and may be rapid, but is at times very slow. The heart sounds feeble; the urine scanty; the ex-



tremities cold; the peripheral circulation poor. There is muscular relaxation, pallor and even cyanosis, the respirations are rapid and irregular. There is fretfulness and restlessness which are rapidly succeeded by semi-stupor. The fontanel is sunken. There may be diarrhea or constipation. The child refuses all food." The temperature is as a rule either normal or subnormal. When the amount of food is small or none is taken for several days the stools are of the starvation type; small, greenish, and often closely resemble meconium. The elimination of fluid, without adequate retention, causes a condition of acute dehydration or anhydremia. These cases rarely survive long enough for the skin to become leathery as in marasmus, but it is often drier than normal. The eyes are sunken and there is a fixed apathetic stare.

*Prognosis and Course.*—The prognosis depends on the severity of onset and the promptness with which treatment is instituted. Many cases apparently dying, if given vigorous treatment respond quickly and recover promptly. Others die within a few days or a week, often in spite of the most careful and energetic treatment. Still others make a slow recovery from the acute symptoms and drag into a state of chronicity which is very difficult to aid. Where congenital deformity exists and is the cause of the inability to take food, little can be done. In the presence of atelectasis some cases can be carried through a critical period, but since the atelectasis is in itself a serious condition, the prognosis is always in doubt. Practically the same can be said for cases of intracranial pressure, when symptoms are severe, although, since we never know from symptoms alone the extent of the hemorrhage, it is well to gavage such infants and keep up the nutrition until the inability to nurse has passed. In such cases a remarkable recovery may be the result.

*Treatment.*—An early recognition and understanding of the condition, together with promptness in instituting treatment are the prerequisites of success. Breast milk is desirable and should be administered whenever available in young infants. The stomach tube should be used without hesitation. Where breast milk cannot be had, whey furnishes an excellent temporary substitute, but since it contains very little nutriment, must not be continued very long. Lactic-acid milk with or without sugar has proved a valuable article of food, not only for the older infant but even for the premature. In fact in my own experience there has not been a single instance where an infant did not digest it well. Where the acute symptoms follow a previous digestive

disturbance, care must be taken not to overcrowd the stomach with too large a quantity of food, but rather should smaller quantities of well diluted food be given until all symptoms except the loss of weight have disappeared. After this the strength of the food may be increased as rapidly as the digestive powers of the infant will permit. We have seen that low temperatures are the rule, but the case of the newly born must be excepted; for the fever occurring during the early days of life may be due to inanition, but is more frequently due to anhydremia. In many instances it is due, however, to infections.

In older infants when the acute symptoms follow digestive disturbances, especially when accompanied by diarrhea, lactic-acid milk has been found to be of great benefit. High protein, given either in the form of lactic acid, skim milk, or skim milk to which has been added curd or other precipitated casein such as the powdered preparations of calcium of commerce (casein), often proves its value in a short time. Some older infants tolerate carbohydrates of the cream of wheat variety very well after a few days of restricted diet; in such instances the gain in weight is much more rapid.

**Anhydremia** (*Anhydremic Intoxication, Water Inanition*).—In the course of diarrhea, and as a result of excessive vomiting, and during a number of other conditions, especially acute inanition and marasmus, the loss of fluid from the tissues of the body may be extreme. The cause is either excessive elimination or failure to ingest or absorb a sufficient amount to maintain a fluid balance. In the course of some acute infections the absorptive power of the intestinal tract is very much interfered with, so that a large proportion of the ingested fluids pass from the bowel in increased amounts, as in diarrhea. There are definite blood changes and the skin and other tissues become inordinately desiccated. This condition of dehydration produces a definite group of symptoms. When in the course of a number of disorders there is an accompanying dehydration it is often difficult to determine whether the symptoms are to be attributed to the original disease or condition or to the anhydremia. This is particularly true of acute inanition and other severe nutritional derangements, as is noted in the discussion of these conditions.

“The symptoms of anhydremia are largely dependent upon a decrease in the volume of the blood.” The most noticeable early symptom is a rapid loss of weight. The face appears drawn, pinched and anxious, and resembles a skull with the skin alone covering it. The

eyes are sunken, as is also the fontanel. The skin over the whole body is dry and flabby, and does not straighten out smoothly when pinched. The tongue is dry as are the lips, and the mouth usually remains open. The urine is scanty and shows albumin in varying amounts and granular casts in abundance. The respirations are deep and labored, with all of the accessory muscles of respiration being brought into play. The pulse is barely perceptible, rapid and irregular. The blood is concentrated and viscid. There may be a subnormal temperature or marked elevation. When treatment is instituted and is effective the temperature promptly subsides unless there is an accompanying infection. Patients affected are at first restless, alert and often excited, until near the end when coma supervenes. Acidosis is often the cause of the fatal ending. This acidosis is due to a lack of excretion of acid phosphates by the kidneys and others produced in the tissues, and not to an actual acid production.

As has been observed dehydration or anhydremia plays a very important part in the symptomatology, and furnishes an indication for urgent treatment. Where vomiting is not a prominent symptom, water should be administered by mouth, either by means of an esophageal tube, drop by drop, or by putting several ounces of water at the time into the stomach at frequent intervals. Where vomiting exists, water is best administered by the intraperitoneal route. A normal saline solution is usually used. This simple procedure is followed by prompt results. It may be repeated as often as indicated, provided the solution is absorbed. Usually six or eight hour intervals are employed. Where symptoms of acidosis are impending a 5 per cent solution of glucose may be substituted for the saline solution. Vomiting constitutes a most serious complication for the administration of food. Where it is protracted, we should rest the stomach completely, and endeavor to combat the dehydration until the infant is able to retain food. The rapidity with which fluids are absorbed, by whatever route administered, is remarkable, and furnishes a fair index to the recuperative power of the child. Hypodermoclysis is hardly advisable since sloughing of the tissues often occurs. Since the temperature is usually low, it is wise to keep the infant warm; the premature jacket often serving well to accomplish this. Blood transfusions, by the longitudinal sinus, in young infants, or in older children by vein or into the peritoneal cavity, often prove to be life-saving procedures. No drugs do good, with the exception of caffeine or camphor, which as stimulants are of definite value.

**Undernutrition in Infancy.**—Between the acute condition—inanition—as already described and the chronic malnutrition of infancy known as marasmus, to be described later, there is a subacute condition of disturbed nutrition which is usually referred to as malnutrition in infants and young children. The term undernutrition probably better describes this condition. It is essentially a disproportion between the supply of food and the demands of the body for fuel and material to build tissue and supply waste. All stages of undernutrition occur from those slightly below average weight to the extreme form known as marasmus.

*Etiology.*—Some of the causes acting to produce acute inanition are also the underlying factors in the less severe and less acute condition under consideration. Congenital or acquired deformities such as pyloric stenosis, harelip and cleft-palate, and stricture of the esophagus resulting from swallowing irritant poisons, are frequent causes. Nursing infants become undernourished because of insufficient supply of breast milk. Artificially fed infants are frequent victims because of an inadequate total quantity of food, or food which is lacking in one or more food elements. Any acute infection which causes the infant to take an insufficient amount of food, or which lowers the digestive powers to an extent interfering with digestion and absorption may be the cause of undernourishment. A common cause of this condition is unintelligent effort to adjust food to the capacity of the infant after minor digestive disturbances, when the statement is made “that nothing seems to agree with the infant, and everything has been tried.” Everything *has* usually been *tried* except the right articles. Individual idiosyncrasy or intolerance for one or another food element accounts for a small number of undernourished cases. In some cases the food has been improperly balanced, one or another element having been in too small quantities, or sometimes one element has been given in excess; this often happens when food too abundant in carbohydrate has been given for a long time, such as in certain proprietary foods, or condensed milk. Perhaps the largest number, however, follow prolonged diarrhea. In this case the digestive power is not only lowered but the diarrhea actually is the cause of a rapid waste of ingested food.

Among more frequent infections causing malnutrition are: Infection of the upper respiratory tract, including bronchitis, tonsillitis, otitis media, and sometimes an unrecognized mastoiditis. Tuberculosis and syphilis should be sought for diligently in all cases which do not gain in a satisfactory manner and when other causes have been



eliminated. Pyelitis often causes a stationary or declining weight and may be a part or sequel of some other infection or may develop without any focus being found. It is entirely likely that absence of vitamins plays a part in determining the state of nutrition in many instances, where the quantity of food appears to be abundant.

*Symptoms.*—The symptoms in undernutrition depend on the age of the infant, the duration of the condition, the underlying cause and a number of other factors. Generally speaking this condition affects infants in the second half of the first year of life and during the second year. In some cases the infant's weight curve fails to maintain a steady increase; at first this is shown in marked daily variations, and is seldom detected unless daily observations are recorded. The weight then becomes stationary, which always means an actual loss in weight in a growing child. Finally an active loss occurs which is quite apparent, and many of these cases are carried to a physician "because they are not thriving."

Growth in length may be arrested; the infant becomes less active, preferring to lie quiet rather than sit up or kick and play in a normal manner. When the condition has lasted for any considerable time the child's strength is perceptibly impaired, and the musculature is definitely under par. This is shown most frequently in the curve of weakness in the lower dorsal spine when in the sitting position, which may be mistaken for a rachitic or tuberculous deformity if the examiner is not careful. This constitutes the only change in posture in these cases.

Instead of the normal sleep of childhood, restlessness is the rule, and when rickets even of a slight degree is present also, the head may sweat during sleep; such sweating is frequently the only sign of the complicating rickets. The subcutaneous fat is perceptibly diminished and the normal turgor of the integument is lost. The skin becomes noticeably drier than normal. The temperature remains normal or becomes slightly subnormal. When the temperature is elevated one should suspect a continuance of some underlying infection as the probable cause of the undernutrition. Because of the low state of resistance susceptibility to disease is increased; especially is this true of infections of the upper respiratory tract, and the child "catches cold" frequently. Although these infants show in many cases no evident disturbance of the digestive tract, in other cases there is either a constant imbalance of digestion or periods of normality alternating with short disturbances. There is no constant bowel manifestation. Consti-



pation is more frequent than diarrhea, and is sometimes obstinate; in other cases a moderate diarrhea is constant, but in such instances the diarrhea is apt to be a manifestation of disturbed digestion or an unbalanced food and is probably the direct cause of the undernourished state. In a few instances of semistarvation, diarrhea may occur. Constipation sometimes alternates with diarrhea. Vomiting practically never occurs except as a sudden and intercurrent complication. When it does occur in the course of undernutrition and is sufficiently prolonged, the case often becomes acute inanition. Some cases show edema, probably due to anemia or to a previous carbohydrate diet.

*Treatment.*—A careful history will often aid in a prompt diagnosis and suggest treatment at once which is suited to the needs of the case. When a thorough and painstaking examination is added to the history the cause of the trouble is more than apt to be discovered in a short while. Treatment, therefore, depends on the previous feeding, the state of digestion at the time of examination, the duration of the condition and to a large extent on the age of the patient. Where an infection is found it should be removed as soon as possible, and if the child appeared to be on a suitable diet no change in the food may be needed, since the removal of the infectious process will be sufficient to bring about an immediate improvement. If the temperature has been elevated and does not return to normal after an attempt to remove infection, it is probable that all infectious foci have not been found. When the history elicits the fact that a single food element has been given in excess, or in insufficient amount, the correction of the error of the single food element may be all that is needed. Where carbohydrate has been given in excess and is withdrawn, and a high protein diet substituted, the child may lose considerable weight in a short time. This is due to the fact that the carbohydrate diet caused water retention and the excess of water has been eliminated. In children under the twelfth month it is best to begin on a diet low in carbohydrate and high in protein; the fat should be kept low also, since there is often difficulty in digesting the fats. In the experience of the author a skim lactic-acid milk has been of great benefit. After the administration of this food for several days, more fat may be added and continued until full milk, acidified, is given. In some cases calcium caseinate, in the form of casec or similar preparations, may be added to the skim lactic-acid milk from the first. The weight should be disregarded, except for accurate records, until the infant is symptom free and we are sure that whatever food is given is being digested; then more and more food may be added, the increases

being made quite slowly, until the child is on a food of sufficient caloric content to meet the requirements of growth. In older children, especially those who have had a diet which was inadequate in total amount, and whose digestion appears not to have suffered materially, the problem is simple, and a well-balanced diet sufficient to the needs of the child may often be administered at once. There is always a water deficiency in undernourished cases and this should be met by copious administration, but resort to the stomach tube or intraperitoneal injections is rarely necessary, though we should never hesitate to use either expedient if the occasion calls for it. In children of any age, after six months, the vitamin content of the food must be attended to. During the first year orange juice should be given early, and in cases which show no digestive derangement, cod-liver oil may be exceedingly helpful. As soon as the digestion is apparently normal after the administration of acid milk or other chosen food, cereals and vegetable purée may be added. In older children fresh vegetables often may be given from the start of the new dietary.

**Marasmus.**—It is quite evident that the several conditions of impaired nutrition in early life, manifested largely by loss of weight, and to which a number of terms are applied, are separated by rather indefinite lines of demarcation and are in reality degrees of the same condition, differing merely in the extent to which the loss of flesh has advanced. Symptoms and signs, however, vary in prominence in the different stages. The chronic, slow developing, and extreme state of malnutrition is known most familiarly as marasmus. "Infantile atrophy" in certain respects better expresses the condition; while "De-komposition," the term used by the German school, leaves much to be desired. Perhaps athrepsia is in fact and etymologically the best descriptive term. Because of its popularity, and its more common association, marasmus will be employed in the present discussion.

A state of extreme malnutrition often occurs in early life in the course of various diseases such as tuberculosis, syphilis, typhoid fever, and in fact any disease or condition in which the waste exceeds the intake or retention or assimilation of food. If for any reason the amount of food is lower than that required by the body the immediate result will be failure to gain in weight. If this deficiency is continued or if a faulty assimilation occurs, the child uses its own body tissues to supply the deficiency, and loss of weight necessarily follows. When loss of weight takes place slowly, steadily and for a sufficiently long period of time a state of extreme malnutrition or marasmus results.

For a time the term *marasmus* was used for cases in which there was apparently an inherent inability to metabolize properly. It is recognized now that there is practically always an underlying cause which can in most instances be found and treated along with the correction of dietary imbalance. *Marasmus* is far more common among the poor, where the food has been inadequate, or in cases where the feeding has been directed by some one ignorant of the needs of the body. Infants and young children forced to live in institutions, where the number of helpers is insufficient to care for a large number of children, are often *marantic*. But even in the best conducted institutions a large number of infants and young children do not thrive and gradually become undernourished, due in large measure to the fact that these children become "hospitalized," for the young human organism needs something more than even the best institution can give, a certain amount of "mothering." In a very large number of children suffering from undernutrition during the second year of life the trouble can readily be traced to improper feeding during the first year. Insanitary surroundings are productive of undernutrition to an extent only exceeded by an insufficient supply of food. There can be no doubt that some children have a feeble or nonresistant constitution, and are prone to numerous infections and disturbance of digestion, these repeated often enough always produce an undernourished state. In general the condition is a secondary one. Many cases are diagnosed as *marasmus* which are found to be tuberculosis.

*Pathology.*—Starvation and dehydration are the outstanding factors which produce characteristic pathological changes in the tissues. It is likely that every organ and tissue participates in both of these processes. The total volume of the blood is diminished as well as its rate of flow. In consequence of this the tissues are poorly supplied with nutriment and in common with the rest of the body the glands furnishing the enzymes suffer, with the result that digestion is impaired, metabolism faulty, and a vicious circle is established.

*Symptoms.*—The condition is insidious in its development, so that in the beginning the appearance may present no evidence. From the onset, therefore, to extreme emaciation, all stages of undernutrition occur. The extreme stage presents a striking and characteristic appearance. The child is so thin that the bony skeleton is plainly visible in every part of the body. The subcutaneous fat is entirely wanting, and the skin is thick and dry, with a leathery feel, hanging in folds and

much wrinkled. The ribs are prominent and the folds of the intestine are visible, the peristaltic waves being clearly seen. The eyes are sunken in the sockets from which the supporting fat has disappeared, but the facial expression is bright and expectant, and never apathetic except in the terminal stage. The cry is a feeble whine, and the child lies in a state of almost complete inactivity. It is literally too weak for any muscular effort whatever. The fontanels are sunken. The



FIG. 47.—INFANT AGED TWO AND ONE-HALF MONTHS, WEIGHING SIX POUNDS THREE OUNCES.

A case of marasmus, due to continual feeding of improper and insufficient food from birth. Marasmus is much more common in later infancy.

color has a grayish hue. The lips are often bright red. The anemia is marked and constant. The temperature is subnormal, and is rarely elevated even in the presence of intercurrent infections, showing a resistance so low that there is no reaction to invading organisms. The pulse is slow and often irregular. The urine is normal but scanty. In some cases a marked edema develops, of which the cause is unknown; some think there is a relation between this sign and the type of food, but such a relation has not been established. A sudden gain in weight in the course of marasmus should make us suspect water retention, and should not be regarded as a sign of improvement. When edema occurs, the chlorids in the urine are diminished or absent, and

when the edema subsides the chlorids again appear. This points to a definite relation to salt retention, but its cause is still not known. Vomiting is rarely present, but hunger is the rule. The infant grasps for anything which looks like food and takes it with avidity. Constant sucking movements are present, and the sucking of a thumb or finger, or more often the whole fist, is common. The abdomen is usually distended, though it may be flat or even scaphoid. Gas accumulation is common, due to poor peristalsis. Constipation is more frequent than



diarrhea, but they may alternate. The accompanying anhydremia accounts for as many of the symptoms as does the starvation. The superficial circulation is poor, and mottling of the skin common. In the terminal stages purpuric spots appear. The lowered resistance permits the development of complications of all kinds at any time. Coryza, otitis, pyelitis, bronchitis and pneumonia are almost regular accompaniments of this condition. Although hunger is the rule, as the case advances and extreme emaciation occurs, the appetite fails and eventually complete anorexia develops. A state of acute inanition may develop in the course of the gradual loss of weight and prove fatal within a few days. In spite of the great emaciation, in which the muscles participate, there is a striking rigidity of the body, due to muscle contraction. The arms and legs are often flexed so rigidly that great force may be required to move the joints. The back muscles are so contracted at times that marked opisthotonos develops, the patient resembling an advanced case of chronic meningitis. Infections of the conjunctiva are frequent and in the later stages erosions of the cornea may occur. This suggests a vitamin deficiency.

*Prognosis.*—The fatality from marasmus is uniformly high under usual conditions. Among the poor and in asylums and other institutions for the care of dependent infants and children a large percentage of the mortality is due to marasmus. The prognosis in individual cases depends on several factors. The age of the patient is perhaps the most important single factor. The younger the infant the more



FIG. 48.—MARASMUS.

Child in the second half of the first year weighs little more than it did at birth. (From Chapin and Royster, *Diseases of Children*.)



uniformly fatal the condition. Conversely the older the patient the better the chance for recovery. The next most important factor is the duration of the condition before treatment is instituted, and closely related thereto is the extent of injury already suffered. It is hardly an overstatement to say that most cases can be saved by intelligent treatment and handling.

*Treatment.*—The same general principles underlie treatment in marasmus as in other grades of undernutrition in young individuals. Infectious processes must be sought for and corrected as promptly as possible. This is far easier in theory than in practice. Some infections are obscure, and in other cases the infant appears unable to overcome the infection while the state of nutrition is impaired. In any event the nutrition must be improved promptly. In infants under a year, one of two courses is to be followed: Lactic-acid whole milk has proved of great value. In many instances marantic infants can be fed an amount of lactic-acid milk equal to their caloric requirements immediately, and are not only not disturbed by a full quota of food, but begin to improve at once. A high protein diet is best tolerated by others. This may be prepared by acidifying skim milk with lactic acid and adding either curd or the case of commerce. In cases where there has been a high percentage of carbohydrate in the diet, this element had better be kept at a minimum until it is shown that there is no great disturbance in the digestive processes. The carbohydrate may then be increased gradually. Some marantic children bear carbohydrate well and it may be given in fairly liberal proportion from the beginning of treatment. Generally speaking, however, it is best to begin the diet with high protein and a caloric value adjusted to the need of the child at its actual weight. Both the carbohydrate and the total calories should then be increased as rapidly as the child will tolerate them until a sufficient quantity to meet the requirements of the child (were it of normal weight for its age or height) is reached. It must always be borne in mind that undernourished children require a larger amount of food per kilogram or pound of weight than normal ones. In older infants the same general precautions should be observed as to beginning low and increasing gradually, but in older children, although we may employ protein milk or lactic-acid whole milk, other articles of diet should be added as soon as it is wise to do so. Marantic children are usually hungry and take food ravenously and care should be taken not to overload the stomach or tax the digestive powers too suddenly or beyond their capacity. When the marasmus has passed to the extreme of weakness, and

anorexia is present, resort must be had to feeding with the stomach tube.

The accompanying water inanition or anhydremia must be attended to very promptly, and is of greatest importance, since these children are often as much in need of fluid as they are of food. Water may be given by mouth, either by frequent libations or by means of the stomach tube, in many cases quite effectively; in extreme cases, however, intravenous or intraperitoneal injections of normal saline solution will be called for and should be administered as often as indicated. Since loss of mineral salts from the body is almost directly in proportion to the loss of flesh, they must be supplied.

Normal salt solution administered as just described will often suffice; in other cases Ringer's solution may be substituted either for the venous injection or for the water given by mouth. Glucose serves a definite purpose in some cases. Intravenous injections are better than intraperitoneal ones, since there is some irritation of the peritoneum by the glucose. Either a 10 or a 20 per cent solution of glucose is injected into the vein, which increases blood volume and furnishes food at the same time, although much of it is excreted in the urine. The combination of glucose and insulin has been found beneficial in aiding in the increase of weight. Marriott recommends for this purpose: "To give insulin, a twenty per cent solution of glucose is prepared and sterilized. Just before the injection, ten units of insulin are added to each one hundred c.c. of the solution and this is injected very slowly into a vein. The volume of solution given is from 20 to 30 c.c. per kilo of body weight. Injections of glucose and insulin may be repeated daily. Insulin should not be given except with 2 Gm. of glucose for each unit of insulin, and should not be given to an infant receiving less than two ounces of sugar per day in the feeding in addition to the amount given intravenously."

Nothing accomplishes so much in extreme degrees of marasmus as blood transfusions. In cases showing any focus of infection which the child is having difficulty in overcoming, whole blood may be used and is probably better. Generally speaking, however, citrated blood serves the purpose well. The amount to be given is usually 20 c.c. per kilogram of body weight or about one ounce for each pound of weight. In some instances intravenous or intrasinus injections are difficult, and usually the services of an expert venupuncturist are required; in such cases intraperitoneal injections prove very satisfactory.

Since the body temperature is often low, artificial heat is required in a number of cases, and should be used promptly when needed. When

practicable, exposure to direct sunlight is of inestimable value; where this is not feasible the quartz lamp may be substituted.

### MALNUTRITION IN OLDER CHILDREN

Malnutrition in infants and young children has been recognized and studied for a long time, and they are now handled quite rationally. The incidence and significance of malnutrition in older children on the other hand have been known and studied only during quite recent years, due to the fact that our attention had not been directed to this phase of child life, largely because children were not studied as a whole, and because no standards existed by which comparisons could be made.

**Signs of Malnutrition.**—It has been observed for a long time by school-teachers and school authorities that a large number of children who appeared to be normal mentally were backward in their classes. When medical examination of school children became general it was found that many of the retarded children hitherto considered healthy, manifested physical defects, and what was more surprising were much undernourished. The combination of physical defects and malnutrition was soon recognized as a definite cause of the retardation. Both parents and family physicians were not only surprised when these facts were discovered and in many instances resented the attitude of school physicians, but opposed to no inconsiderable extent the efforts made to correct the abnormal conditions. The almost universal argument was that many of the children found suffering from abnormal conditions had not been sick for years, and that it was strange that they should develop such conditions just as soon as they entered school. The fact was, of course, that they did not develop these conditions suddenly, but that they had developed them gradually during the period between infancy and the entrance to school. The fact that no acute illness had occurred had led to the false assumption that the children were normal. They had no systematic examination during this time and even if they had been acutely ill, they had been examined only to an extent made necessary by the definite illness then present. A further cause for the failure to discover undernutrition was that no standards existed by which to make comparisons.

A second factor in the recognition of malnutrition was the examination of recruits for the army during the World War, when a large per cent was found to be sufficiently undernourished to be undesirable for

taken from among the author's own cases, with the exception of two or three, whose posture suited the purpose better than those in his possession.

**Etiology.**—The main and precipitating cause of malnutrition is food which is insufficient in quantity, inadequate in quality, or improperly assimilated. There are a number of secondary causes, all of which bear directly on the primary cause.

*Insufficient Food.*—A definite number of children of the poorer classes are undernourished because of an insufficient amount of food. Food may be insufficient also when the child has for any reason a poor appetite and takes very little.

*Inadequate Food.*—This term is used to designate food which may be sufficient in total quantity but is of a quality not suited to the needs of a growing child. It is an unbalanced diet. It may be high in carbohydrates and insufficient in protein, and this constitutes the most frequent imbalance in the food of older children. There may be sufficient total quantity, sufficient protein and yet the vitamins be lacking.

*Faulty Food Habits.*—One of the most important causes of malnutrition, and the one almost invariably found in the course of routine history taking, is faulty food habits. Among the most important of these is the eating of large quantities of sweets. Children are often given excessive amounts of sweets, either by adding sugar to all food to such an extent that they will eventually take only food that has been sweetened, or by allowing them to eat large amounts of candy between meals. Irregular meal hours; meals hurried through by the child to get out to play or to reach school on time, and eating between meals are faulty habits frequently found. The last may not be wrong from a physiological standpoint, but is certainly very wrong from the standpoint of food habits. At least psychologically it is wrong.

*Faulty Health Habits.*—Second only to faulty food habits are faulty health habits. These include insufficient sleep at night, the lack of a regular rest period during the day, neglect of daily evacuation of the bowels at a regular hour, sleeping in a room in which there is a lack of fresh air, and sleeping with another.

*Physical Defects.*—Many physical defects bear directly on the state of nutrition. Decayed teeth, usually painful, prevent proper mastication of food. The bad breath caused by decayed teeth is noticeable to the child itself in many cases and is sufficient to impair the appetite. Diseased tonsils furnish a constant supply of toxin absorbed by the body which prevents the proper assimilation of food. Exuberant



adenoid tissue prevents proper aëration of the body and also causes frequent infections of the upper respiratory tract. Pyelitis, otitis media, sinusitis and any other focus of infection may be a determining cause. Gonorrheal vulvovaginitis is an overlooked and a very frequent source of focal infection, and causes malnutrition.

*Home Condition.*—That “as the home is conducted so the child conducts itself,” is an almost invariable observation, and has an important bearing on the child’s general condition. Lack of home control is one of the most important of the many causes contributing to a state of malnutrition. It has been stated that malnutrition is common in the children of parents who are not strong or who have some such disease as tuberculosis or syphilis. There appears, however, to be no direct relation between the child’s nutrition and tuberculosis in a parent. Even in the young infant it is a remarkable fact that some of the best developed and nourished children are born of mothers who have tuberculosis and who are themselves poorly nourished. Unless the infant itself acquires the disease at an early age, it is devoid of influence. The offspring of syphilitics, when young, are frequently undernourished, but only when they are themselves subjects of the infection. In all young infants who do not thrive and in whom no other cause for the undernourishment can be found, syphilis should be looked for. The malnourished children of neurotic parents, who are nervous themselves, are often said to inherit a nervous temperament from one or the other parent. This may be so, and can hardly be disproved, but it is far more likely that the children reflect the conduct of those around them and acquire nervous habits, rather than inherit them. Children who live in an atmosphere where tension prevails, and who are alternately nagged and overindulged, will reflect such handling in nervous manifestations. Children naturally love to be the center of attraction, and will often refrain from eating in order to hear a mother tell a friend that the child “just will not eat.” Children who are not made to mind and to cultivate a calm demeanor, but who are allowed to have their own way, are especially prone to malnutrition. Many children are allowed to choose their food rather than have it chosen for them, with the result that they almost invariably choose sweet foods, and not those which build tissue, and hence become undernourished. Children are allowed to eat between meals, usually choosing candy and other sweets, thus preventing a healthy appetite for the desirable articles of diet. Among influences which are detrimental, and which are perhaps for convenience classed with home influences, may be mentioned over-



loading children with work out of school hours. Such work is regarded as acquiring certain accomplishments, such as dancing, music and the like, quite desirable in themselves, but usually taught at a time when the child had better be out of doors engaged in health-promoting sports.

Some discussion has taken place as to the extent that intestinal parasites interfere with nutrition. There is no doubt in the opinion of the author that they do play a part in malnutrition, in some instances an apparently important part. Hookworm infestation is a well recognized cause of even extreme malnutrition; strongyloides infestation and some others of the same type may be as detrimental as hookworm. Lumbricoid worms often interfere with nutrition to a marked degree, and it seems entirely likely that any infestation may be of etiological importance.

*Treatment.*—A carefully taken history, carefully considered in relation to the condition of the child, is a prerequisite to success in treating malnutrition. The history should include, as far as possible, the progress of the individual from infancy to date, as regards weight at varying intervals and what the food was at each of these periods. Each illness should be recorded and the time of its occurrence; especially should this include the number of attacks of sore throat. These facts are not always easy to ascertain, since the memory of people is short in regard to such matters, and this is even true with the fondest and most observing parent. A careful record of the child's progress in school should be made, for this often tells of fatigue.

Next in importance is a careful physical examination, which of course should include especially age, height and weight. In addition to these, of special importance are the lungs, heart, tonsils and adenoids and teeth. Pyelitis and vulvovaginitis should be searched for diligently.

Physical defects should be corrected as soon as possible, since it is difficult and often impossible for a child to gain in weight regardless of the quantity and quality of food as long as a physical defect, which may be the primary cause of the undernutrition, remains as a continued source of infection or a handicap in other respects. By removing the defects the child is made "free to gain" in the words of W. R. P. Emerson. Decayed teeth should receive immediate attention, and the only safe rule to follow is that a tooth is either good enough to be filled or bad enough to be extracted. Malocclusion should be corrected wherever practicable. Perhaps the most serious of all infections is that of the tonsils. Few normal tonsils are found in the course of routine examinations of large numbers of children. In many instances

where a child is malnourished and whose food appears to be adequate, and the child is securing enough rest, there is a definite improvement almost immediately on the removal of diseased tonsils. One rule must be insisted on, however, and that is that all children should remain in bed at least five days, and preferably a week after the operation. The reason is quite definite. The child takes only a small amount of food for several days after the operation and the rest is needed, so that this small amount of food will more nearly approximate the requirement; in this way unnecessary loss of weight may be prevented. Where the malnutrition is apparently due to a suspected or actually diagnosed tuberculosis, it is evident that treatment must be directed to that condition at once. Any heart lesion calls for special handling, not within the province of this book to describe. The total removal of all physical defects before specific treatment directed to the malnutrition is instituted, is imperative.

Second in importance to the above is to secure as prompt and as whole-hearted coöperation from the parents as possible. Without this aid failure results in the vast majority of cases. The parents must be shown the significance of this condition not only as regards the present health of the child, but, more important still, the effect on its whole future. Parents must be convinced that it is wisest to be firm with the child and insist on the prescribed routine, and be shown that yielding in the smallest detail will vitiate almost every effort made in the child's behalf. Parents must be shown the value of food and what the relations of the various forms of food are to the child's economy. This must and can be done in simple and nontechnical language, so that even a relatively ignorant mother can understand.

Next in importance is sufficient rest for the child. This means a definite number of hours' rest at night in bed, in accordance with the needs stated elsewhere in this volume. Besides the regular sleep at night, there must be rest periods during the day. There may be one rest period of one to one and a half hours in the middle of the day, or two periods of an hour each twice a day; in the middle of the morning and in the middle of the afternoon, according to the needs of the child, varying with the amount of malnutrition and the degree of nervousness. In severe cases of either undernutrition or marked nervousness, it is best to put the child in bed all of the time for from one to six weeks, depending on how rapidly weight is added and symptoms disappear.

In many instances where the weight is very low or nervous symptoms are marked it is necessary to remove the child from school, and

in any case both mental and physical activities usually have to be reduced to a minimum for awhile. This calls, in most cases, for the interruption of dancing lessons, music lessons and other "refined accomplishments" which interfere with rest or recreation.

The child should sleep alone, in a well ventilated room, preferably on a sleeping porch; where the rest period is needed in the daytime it is better that this be taken in a quiet, darkened, but well ventilated room free from interruptions. Exercise should be limited for a while to quiet walking, and competitive games and running exercises stopped.

The food must receive the most careful consideration. It must be well balanced in quality and sufficient in quantity. The diet lists given in the previous chapter may serve as guides, and the quantity must be adequate in total calories to meet all requirements. The caloric needs for the various age periods are also given on page 125. Of as much importance as the quality and quantity of food are proper food habits. By this is meant that the meals should be at regular and definite intervals. The best hours are breakfast at eight o'clock, dinner at one and supper at six. Nothing should be taken between meals except when it is difficult to induce a child to take a sufficient amount at the regular hours; in such instances it may be found necessary to give a small meal of a few crackers and a glass of milk between the regular meals. This should not be kept up for very long, since the best way to induce a healthy appetite is through an empty stomach. A child should be taught to eat what is put before it, and should be made to remain at the table for at least twenty minutes. If it does not eat the meal it should be allowed to leave the table, but should be allowed no food again until the next meal time. A few days of this discipline will usually form a good habit of eating. All directions as to diet should be written for the guidance of the mother, and a record of the food taken should be kept. A convenient method of doing this is by means of the record books of the health clinics of Boston.

In the accompanying table of food values no claim to absolute accuracy is made, convenience for a mother being considered. It will be found, however, that an overestimate of food value in one instance is counterbalanced by an underestimate in another instance. In this way the average for the twenty-four hours approximates accuracy closely enough to meet the needs of the situation. The second column represents the amount of the article of food required to furnish 100 calories. The third column shows the number of calories of each food element represented in that portion.

# QUANTITIES OF FOOD NECESSARY TO YIELD 100 CALORIES, WITH THE PROPORTION OF PROTEIN, FAT AND CARBOHYDRATE \*

ts = teaspoonful    tbsp = tablespoonful    h = heaping    aver = average

FISH AND MEAT			Calories		SOUPS			Calories	
	100 Cal.	Oz.	P.	F. Ch.			Oz.	P. F. Ch.	
Cod, broiled	3 oz = size 3 chops	3.6	90	10-0	Cream—	30 cal. to the oz.	3.9	12-70-18	
Haddock, broiled		3.3	90	10-0	Asparagus		3.8	10-73-17	
Bluefish, broiled		2.4	71	29-0	Celery		3.2	12-43-45	
Halibut, broiled		3.	61	39-0	Corn		3.2	16-48-36	
Mackerel, broiled	2 oz = size 2 chops	2.6	56	44-0	Pea		20 cal. to the oz.	3.5	10-70-20
Salmon, canned		1.8	45	55-0	Tomato	3.8		20-38-42	
					Clam chowder	3.9		34-35-31	
					Fish chowder				
Roast veal	1 oz = size 1 chop	2.7	71	29-0	Thick—	4 cal. to the oz.		5.4	20-20-60
Roast chicken		1.9	73	23-4	Bean		6.	72-12-16	
Dried beef		1.7	67	33-0	Chicken		6.	26-2-72	
Boiled mutton		2.1	74	26-0	Split pea		4.3	23-49-28	
Round steak, broiled		1.9	48	52-0	Meat stew		5.	23-57-20	
Roast pork	1 oz = size 1 chop	1.7	55	45-0	Oyster stew	4 cal. to the oz.	32.	84-8-8	
Tripe		2.4	46	54-0	Clear—		29.	85-0-155	
Roast lamb		1.8	41	59-0	Bouillon		25.	85-0-15	
					Consommé				
					Vegetable				
Tenderloin steak	1 oz = size 1 chop	1.3	34	66-0	VEGETABLES				
Roast mutton		1.1	33	67-0	Asparagus				
Ham, boiled		1.2	29	71-0	fresh	20 stalks	15.9	32-8-60	
Corned beef, boiled		1.2	21	79-0	cooked	7 h tbsp	7.	34-6-60	
Tongue		1.2	27	73-0	Beans				
Roast beef	1 oz = size 1 chop	1.2	46	54-0	baked	1 tbsp	2.7	21-18-61	
Lamb chop		1.	40	60-0	lima, fresh	2 tbsp	4.4	21-4-75	
Roast turkey		1.	40	60-0	string	10 tbsp	8.5	22-7-71	
Roast duck		1.	30	70-0	Beets	6 h tbsp	7.7	14-2-84	
					Cabbage	60 h tbsp	11.	20-8-72	
Sausage	two-thirds	.7	20	78-2	Carrots	4 h tbsp	5.8	10-5-85	
Bacon	1 slice	.5	13	87-0	Cauliflower	24 h tbsp	11.5	23-15-62	
Salt Pork	1 in sq	.5	4	96-0	Celery	1 bunch	19.	24-5-71	
Clams	12 to 16	4.7	56	8-36	Corn				
Oysters	twelve	7.	49	22-29	canned	2 h tbsp	3.5	11-11-78	
Sardines	four	1.3	34	66-0	green	2 ears	3.5	13-10-77	
Lobster	3 h tbsp	4.1	78	20-2	Cucumber	2 large	20.	18-10-72	
Scallops	2 h tbsp	2.5	80	1-19	Lettuce	2 large			
					heads	18.	25	14-61	
					Mushrooms	8 large	7.6	31-8-61	
					Onions, raw	4 h tbsp	7.2	13-6-81	
					Parsnips	5 h tbsp	5.8	10-7-83	
					Peas				
					green	4 h tbsp	3.5	28-4-68	
					canned	4 h tbsp	4.4	25-3-72	
					Potatoes				
					sweet, baked	½ aver	3.	6-5-89	
					white, baked	1 aver	3.	11-1-88	
					Rhubarb				
					stewed	2 h tbsp	1.7	1-2-97	
					Spinach, boiled	4 h tbsp	21.	12-8-80	
					Squash	4 h tbsp	7.4	12-5-83	
					Succotash	3 h tbsp	3.5	15-9-76	
					Tomatoes				
					canned	12 h tbsp	15.6	21-8-71	
					fresh	4 aver	15.5	16-16-68	
					Turnips	6 h tbsp	8.7	13-4-83	

Lean part lamb chop weighs 1 oz.

Fish and meat vary in value according to fat present.

DAIRY PRODUCTS			
Butter	1 pat	.4	1-99-0
Cheese			
American	1 cu in	.9	25-73-2
Cottage	2 h tbsp	.1	76-8-16
Cream, full	1 cu in	.9	25-73-2
Neufchâtel	1 cu in	.9	22-76-2
Pineapple	1 cu in	.9	25-73-2
Roquefort	1 cu in	.9	25-73-2
Swiss	1 cu in	.9	25-74-1

\* These analyses are based for the greater part upon Bulletin 28, Office of Experiment Stations, U. S. Department of Agriculture. The values of cooked foods are necessarily approximate and allowance should be made for dressings, sauces, etc., especially the fat in which foods are cooked. (After Emerson).

## CEREALS

		Calories	
		Oz.	P. F. Ch.
Corn flakes	10 h tbsp	1.	6- 4-90
Cream of wheat	4 h tbsp	6.	12- 3-85
Farina	4 h tbsp	6.	12- 4-84
Grape nuts	2 h tbsp	1.	13- 2-85
Hominy	3 h tbsp	4.2	11- 2-87
Indian meal	3 h tbsp	6.	10- 5-85
Macaroni, boiled	4 h tbsp	4.	15- 2-83
Oatmeal	4 h tbsp	5.6	17-16-67
Puffed rice	10 h tbsp	1.	9- 1-90
Rice, boiled	4 tbsp	3.1	10- 1-89
Shredded wheat	one	.9	13- 5-82

## BREAD

White	3×3½×1 in	1.3	14- 6-80
Whole wheat	2½×2½×½ in	1.4	16- 3-81
Corn	2×2×1 in	1.2	10-24-66
Biscuit	one	1.3	11-27-62
Roll, Vienna	one	1.3	12- 7-81
Zwiebach	3 pieces	.8	9-21-70
Pilot	¾ cracker	.9	11-12-77

## CRACKERS

Boston	one	.9	11-19-70
Educator	twelve	1.	40- 3-57
Graham	two	.8	9-20-71
Oatmeal	seven	.8	11-24-65
Oyster	twenty-four	.8	7-24-69
Saltines	six	.8	10-26-64
Uneddas	four	.9	9-20-71

## FRUITS (FRESH)

Apple	1 large	7.3	3- 7-90
Banana	1 large	5.5	5- 5-90
Blackberries	4 h tbsp	6.1	9-16-75
Cantaloupe	one-half	8.6	6- 0-94
Grapefruit	one-half	11.4	3-12-85
Grapes, Concord	1 bunch	4.8	5-15-80
Lemons	1 large	7.6	9-14-77
Orange	1 large	9.4	7- 2-91
Peach	3 aver	10.5	6- 3-91
Pear	1 large	6.3	4- 7-89
Pineapple	2 slices	8.2	4- 6-90
Raspberries	9 h tbsp	5.3	10-14-76
Strawberries	10 h tbsp	9.	10-15-75
Watermelon	.....	11.7	5- 6-89

FRUITS (DRIED)  
(Edible portion)

Dates	3 large	1.	2- 7-91
Figs	1 large	1.1	5- 0-95
Prunes	3 large	1.4	3- 0-97
Raisins	10 large	1.1	3- 9-88

## DESSERTS

		Calories	
		Oz.	P. F. Ch.
Cakes			
Sponge	2×2×1 in	.9	11-19-70
Choc. layer	2×1½×1 in	1.	7-22-71
Frosted	2×1½×1 in	1.	6-22-72
Gingerbread	2×2×1 in	1.2	8-22-70
Lady fingers	two	.9	10-12-78
Macaroons	two	.8	6-33-61
Cookies	two	.8	7-22-71
Choc. éclair	½ small	.8	4-33-63
Doughnuts	½	.8	6-45-49
Pies			
Custard	½ of a quar	1.9	9-32-59
Lemon	½ of a quar	1.4	6-36-58
Squash	½ of a quar	1.9	10-25-65
Apple	½ of a quar	1.6	3-41-56
Mince	½ of a quar	1.2	8-38-54
Puddings			
Bread	1 h tbsp	1.6	10-20-70
Baked custard	2 h tbsp	2.6	17-37-46
Rice custard	2 h tbsp	2.7	8-13-79
Apple tapioca	2 h tbsp	3.	1- 1-98
Indian	1 h tbsp	2.	12-25-63
Ice Cream	1 h tbsp	2.	6-55-39

## SWEETS

Cocoa	4 h ts	.7	17-53-30
Chocolate	½ sq	.56	8-72-20
Fruit sauces	2 tbsp	2.	1- 3-96
Jellies, all	1 tbsp	1.	1- 0-99
Marmalade	1 tbsp	1.	1- 2-97
Honey	1 tbsp	1.	1- 0-99
Sugar			
granulated	4 ts	.9	0-0-100
powdered	4 h ts	.9	0-0-100
cube	4 lumps	.9	0-0-100
domino	6 small or		
maple	3 large	.9	0-0-100
Maple syrup	4 ts	1.	1-0- 99
	1 tbsp	1.2	0-0-100

## NUTS

Almonds	eight	.5	13-77-10
Brazil	three	.5	10-86- 4
Chestnuts, Italian	seven	1.5	10-20-70
Filberts	ten	.5	9-84- 7
Peanuts	13 double	.6	20-63-17
Pecans	eight	.5	6-87- 7
Walnuts, English	ten	.5	10-83- 7

## MISCELLANEOUS

Olives, green	seven	1.6	1-84-15
Alcohol		.5	- -



## FOODS USED FOR INFANTS AND IN ILLNESS

	Cal. to oz.	oz.	Calories P. F. Ch.		Cal. to oz.	oz.	Calories P. F. Ch.
Albumin water				Top milk 7%	27.	3.75	15-66-19
1 white to 8 oz.	3.5	28.	100- 0-0	(3.5 7 4.5)			
Barley water				Skimmed milk	14.	7.	30-33-37
1 oz. to qt.				(3.6 1.8 4.5)			
(.13 .07 2.44)	3.1	32.	4- 6-90	Buttermilk	11.	9.	41-13-46
Barley gruel				(3.6 .5 4.06)			
2 oz. to qt.				Condensed milk			
(.27 .15 4.89)	6.2	16.	4- 6-90	(Eagle brand)			
Rolled oats water				(8.43 6.94 50.69)	100.	1.	11-20-69
1 oz. to qt.				Six parts water			
(.26 .14 1.67)	2.5	40.	12-14-74	(1.20 .99 7.23)	13.	7.5	11-20-69
Rolled oats gruel				Nine parts water			
2 oz. to qt.				(.84 .69 5.1)	9.5	10.75	11-20-69
(.52 .28 3.34)	5.	20.	12-14-74	Whey			
Beef broth	1.1	88.	100- 0-0	From whole milk	10.5	9.5	11-25-64
Chicken broth	1.	100.	30-55-15	(.94 .96 5.49)			
Beef juice				Eiweismilch	12.	8.5	30-56-14
cold process	14.	7.	100- 0-0	(13-25-15)			
warm process	19.	5.3	78-22-0	Koumyss			
Orange juice	14.	1.	0-0-100	From cow's milk	12.5	8.	24-38-38
Olive oil	250.	.4	0-100-0	(2.66 1.83 4.09)			
Malt soup (Keller's)	20.	5.	12-16-72	Sugar, gran 4 ts		.86	0-0-100
(12 1.2 12.1)				powdered 4 h ts		.86	0-0-100
Human milk				Milk sugar		.9	0-0-100
(1.25 3.5 7.0)	20.	5.	8-52-40	Dextrimaltose 3 h ts		.9	0-0-100
Cow's milk				Mellen's food 3 h ts		.9	12- 6-82
(3.5 4.0 4.5)	20.	5.	20-52-28	Malted milk 3 h ts		.83	15-19-66
Rich milk				Wheat or barley flour	1.	12- 3-85	
(3.5 5.0 4.5)	22.	4.5	18-59-23	Apple sauce 2 tbsp	2.2	1- 4-95	
Cream				Prune sauce 3 med w juice	3.8	2- 1-97	
Top milk 40%	100.	1.	2-95-3	Scraped beef	2.	61-39-0	
(2.2 40 3)				Egg one large	2.1	36-64-0	
Top milk 16%	50.	2.	7-84-9	white seven	6.4	97- 3-0	
(3.25 16 4.05)				yolk two	.94	17-83-0	
				Zwiebach three	.8	9-21-70	

The amount of food at each meal is to be regarded; the calories calculated from the preceding table and a total for the day computed. The number of calories the child should take should be prescribed by the physician, and the mother or attendant should see that the child approximates this number as closely as possible in its daily food. It is comparatively easy to vary this by very slight calculation. For example, one slice of bacon adds 100 calories, likewise one pat of butter adds 100 calories. In this way it is easy to regulate with a fair degree of accuracy the actual number of calories taken in the twenty-four hours. An example of how this can be done follows:

## SUGGESTED DAY'S DIET LIST

TIME	FOOD	CALORIES	NOTES
7.30 A. M.	BREAKFAST		
	4 tablespoons oatmeal .....	100	
	2 oz. cream .....	100	
	2 teaspoons sugar .....	50	
	1 egg .....	100	
	1 slice toast .....	100	
	1 pat butter .....	100	
	3 large prunes (with juice) .....	100	
	5 oz. cocoa (made with milk) .....	125	
	1 teaspoon sugar .....	25	
		800	
10.15 A. M.	LUNCH		
	Milk, 1 glass .....	150	
	2 large graham crackers .....	100	
		250	
12.30 P. M.	DINNER		
	1 medium baked potato .....	100	
	1 pat butter .....	100	
	1½ oz. meat or fish .....	150	
	2 tablespoons carrots or		
	2 tablespoons spinach .....	50	
	1½ slices of bread .....	150	
	1½ pats butter .....	150	
	3 tablespoons rice pudding .....	150	
		850	
3.45 P. M.	LUNCH		
	1 bread and butter sandwich .....	150	
	1 large apple or orange .....	100	
		250	
6.15 P. M.	SUPPER		
	2 slices milk toast (8 oz. milk, 1 pat butter) .	450	
	2 tablespoons apple sauce .....	100	
	1 molasses cookie .....	50	
		600	
	TOTAL .....	2750	

DRINK AT LEAST 3 GLASSES WATER DAILY

Not only should a record of the articles of food taken be kept, but the amounts also, in household measures, together with their caloric equivalent. In this way only can the needs of the child be arrived at, and also only thus can a sufficiency of food be insured. A sample page from the food record book alluded to is shown. In many instances the child takes pride in keeping its own record, and this is desirable because in the case of older children the coöperation of the child is as

important as that of the parent. When a child once understands what it is all about success is sure. There has been much debate as to the best manner in which to accomplish results. In private practice, work must be done on the individual child and with the individual mother. In school work and in children's clinics, it is often necessary to hold nutrition classes. In these, a group of children is instructed together and records kept and shown. In some instances the parents are present at the weekly weighings and observations; in some places it has been found more advantageous to have this work done without the presence of the parent, in which instance the pride of the child in a health competition is aroused and the results are often excellent. The interest of the teacher is valuable and aids in the progress of the case. It is to be hoped that the acquiring of a high standard of health will be credited in the curriculum of all schools, in the not distant future.

We have shown that a faulty posture was one of the most striking manifestations of malnutrition. What of its correction? In extreme cases this phase of the work belongs in the realm of orthopedics. Setting up exercises as usually practiced do little or no good. A trained physical culturist, possessing a thorough knowledge of anatomy is in the best position to accomplish results. No muscle correction should be attempted, however, until the child has gained weight at least up to the average for height, and preferably to 10 per cent beyond the average. Two things should be done in this direction, however, while the treatment of the malnutrition is being conducted. The child should be taught to sleep on the back without a pillow under its head. This is hard at first but will be found to be quite comfortable after a few nights. After this habit is formed a small firm pillow should be placed under the back between the shoulders. In this way the weight of the shoulder girdle and arms will overcome much of the tendency to round shoulders.

Since even the slightest infection with tuberculosis may be the main cause of a malnourished state, it would appear wise to discuss very briefly the dietary treatment of this disease, without encroaching on the scope of another of this series of volumes. There has been much discussion pro and con regarding the kind of food needed by the tuberculous. The part played by calcium in the food particularly as influencing the healing process in the lung, has received much attention from one and another observer. Some of this observation has resulted from the calcareous glands found in proximity to the bronchial tree, and supposed to be an evidence of healing. Since fat was supposed to favor the deposition of calcium it was stressed in the diet to a consider-

able extent. That cod-liver oil promotes the retention of calcium is well known now, but this is due to the influence of the contained vitamin and not to the fat. That fat in the diet in liberal proportions tends to prevent infections and especially tuberculous infection is believed by many, though of this there is no proof. On account of all these ideas the administration of cod-liver oil in large amounts, often to the detriment of the digestion, was in general vogue for many years, so much so that fat cures by means of certain emulsions became the fashion and endured for a long time. The vitamin in cod-liver oil does promote retention of calcium especially in rickets and perhaps other conditions where the calcium in the blood serum is below normal, but there is no evidence that the serum calcium can be increased beyond the normal by any such administration.

Forced feeding carried to a high degree of forcing also came in for its share of popular attention, with the result that overfat individuals without stamina were seen with digestive systems impaired to an extent that they were soon unable to take enough food to keep them at a reasonable state of weight. Forced feeding has no place in the treatment of tuberculosis, particularly in children, since it is very apt to disturb the digestive powers and cause derangements which are difficult to overcome if indeed they are ever overcome.

The problem of the cure of tuberculosis in children is essentially that of the undernourished child, and the principles of feeding in an effort to remedy an advanced state of malnutrition as laid down in the section devoted to that condition apply with equal force to this specific disease. The main indications in feeding an undernourished child are, as will be recalled: to maintain life, to promote growth, to meet the katabolic waste, and to meet the needs of activity. To these must be added, in treating tuberculosis, the overcoming of an acute infection, which is especially wasting in its nature. A balanced ration is of as great importance in this disease as it is in all other conditions of life. Variety is not essential but very desirable, in order to prevent the patient's tiring of all food. Milk, eggs, leafy vegetables, and fresh fruit constitute a balanced diet, the main reliance being placed on milk and eggs. When the requirements of growth are met by an adequate supply of protein, the needed additional calories may be supplied by carbohydrates. In the feeding of the tuberculous child a sufficient amount of protein to meet the requirements of growth must be supplied; the fat requirement will usually be met by the usual articles of food, the additional calories may be supplied by cereals and bread.

The main indication therefore is that the food should be given in measured amounts, and not merely by guess, and a careful record kept of all food given; not with the idea of forcing a large amount, but that we may know at all times whether the child is getting a sufficient amount of food to meet all requirements.

When a child showing a definite infection with tuberculosis is first placed on a cure the weight for the height should be recorded and the child placed at complete rest. The food should then be prescribed in amounts sufficient to maintain that body weight without loss. Food should then be slowly increased until an amount sufficient for every need of the child at the weight it should be is being taken. If this is well tolerated, and no vomiting or other evidence of digestive disturbance occurs, and the appetite keeps up, the amount of food should be increased gradually until the child is taking a somewhat larger amount than is actually needed for a child of normal weight. This does not constitute forcing, and may not be needed in every case.

The complete rest will enable the child to put on more weight on a smaller amount of food than one who is taking even a small amount of exercise. The rest, complete or partly modified, should be maintained until the child is above the average weight for its height. The author prefers, where practicable, to have the child reach a weight which is 10 per cent beyond the average before permitting much activity. The reason for this is that just as soon as exercise is begun more food in proportion to the weight is required and there will be a loss of weight, which varies with circumstances and with the individual child. The object is to maintain a weight in keeping with the average for the height and to maintain this level as the child increases in height.

It will be seen therefore that the dietetic treatment of tuberculosis differs in no essential from that employed in any undernourished child. A point too often forgotten by some who treat tuberculosis exclusively is that the patient and not the disease should be treated and that the tuberculous are not only just as apt to have intercurrent infections and other ailments, but are unfortunately more prone to them. All intercurrent infections should be corrected promptly and carefully, since they not only prolong the process of arrest of the tuberculous infection, but as in all other cases impair the digestion. A sound digestive system, functioning smoothly, is the sheet anchor in the care of the tuberculous child.



## CHAPTER XI

### EFFECTS OF INANITION AND MALNUTRITION

Malnutrition and inanition<sup>1</sup> have been studied fairly extensively in their relation to immediate effects; that is to what extent an interruption of normal food ingestion or administration may occur, and the individual survive. Such studies have embraced acute inanition, malnutrition, especially the less acute and fairly prolonged cases, and the undernutrition of the older child. Practically all of these observations have been made with the chance for immediate recovery in mind. Little or no thought has been given to the ultimate results of a restriction of food, either partial or complete, on the future development of the individual. Aside from the immediate outcome, as affecting the mortality rate, such effects are more important from the standpoint of the ultimate results than the immediate. As a result of any form or degree of malnutrition, undernutrition, or inanition, must be asked the question, What is the adult product going to be? Is the individual who has been deprived of adequate nutrient elements at a critical period of growth and development going to develop into a sturdy adult, able in all respects to carry on as a strictly normal individual? And again, and what is in a sense a far more important question: Is an individual who has been made to suffer, physically, from a deprivation of food, either qualitatively or quantitatively, going to be able as an adult to propagate normal and virile offspring?

All of these questions are important and have a far-reaching effect on the development of the race. It is unfortunate that they cannot be answered finally at this time. Many difficulties lie in the way of investigations on human beings, which might throw light on the subject and enable us to correct errors which are undoubtedly being made daily. Records are poorly kept, if kept at all, and the opportunity to follow any large number of children from birth to maturity is rarely afforded: so in large measure, we must rely for an answer to all of these questions, on animal experimentation and their analogy to our own clinical observations on children.

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<sup>1</sup> A full account of experimental evidence of the effects of starvation is presented by Jackson, *The Effects of Inanition and Malnutrition upon Growth and Structure*, P. Blakiston's Son & Co.

Inanition actually means complete and absolute restriction of all food, in other words inanition usually refers to what we are accustomed to know as starvation. The term inanition is often used, however, to apply to complete food or water restriction, or to both, and sometimes to mean partial restriction in either or both. In the case of food there may be still further qualification of the meaning of the term so as to apply to either quantitative or qualitative restriction or to both. Thus while inanition refers to starvation, a number of other terms such as malnutrition and undernutrition are in common use to signify all kinds of variation in food restriction or deprivation. A number of terms applied to the various degrees of food inadequacy have already been employed in this volume; the shade of meaning differing to some extent according to the nature of the disorder or the type of observation under discussion.

All forms of life are affected by restriction of food, be this restriction in what is generally called the food elements, or in water alone, or in both. Some of these effects are temporary, the body recovering apparently completely when a return to an adequate supply is made. In some cases the effect is permanent, and either stunting or arrest of development occurs in the organism as a whole or in various parts of the organism; in either event a deformity is the result. Studies, varying in thoroughness, have been made on plants, invertebrates, and vertebrates, including the higher animals and to a lesser extent, man. That a permanent effect is produced in many instances is proven in practically all observation on all but man, and in this case there is no doubt that such results are in strict analogy to those in the lower forms of life, though absolute proof is still lacking.

Plants are peculiarly susceptible to environmental influences, probably to a greater extent than animals. This susceptibility applies particularly to the food supply. Inanition in plants restricts or inhibits growth, during the developmental period, causing either a general arrest of growth and development, or variations of both form and structure, which differ materially from the normal. In some cases there occur both an arrest of development and change in form.

After the period known as the developmental period is passed, plants are less susceptible to deprivation of food, but even at the later stage, if such influences continue to act, or are inaugurated at this stage, certain atrophic changes degenerative in their nature occur, particularly in the protoplasm. Such changes occur whether the inanition is partial or complete, and even perhaps when one or two

essential elements are wanting in the food. In addition to the generalized effects which are produced by partial or total lack of food, from a quantitative standpoint, there are definite changes, or partial arrest, resulting from the absence from the diet of one or another food element. These results are specific for the element involved. For example calcium is necessary to the formation of cellulose, and without this element cellulose is not formed. Both potassium and phosphorus are necessary to mitosis, and when the plant is deprived of these elements no mitosis occurs. In plants even the determination of sex is dependent on the presence in their diet of certain mineral salts.

Some of these effects are obviously peculiarly characteristic of the plant kingdom; but they closely resemble those observed in animals as well, the analogy often being very striking. It requires but a little thought for us to realize that various circumstances determine what the effect of inanition will be on any living organism. The two most potent factors in this determination are the time (development) at which the organism is subjected to food deprivation, and the duration of the deprivation. We have already seen that starvation in plants affects the size of the plant permanently; and that morphological peculiarities are also produced and that sex may be altered. These characteristics are environmental in origin and not heritable, though inheritance of variations produced by starvation has been observed. Whether this inheritance is temporary, that is, only for one or more generations, or is permanent, has not been demonstrated. Various plants can withstand starvation and semistarvation for varying periods of time without dying; and also in fact without the occurrence of developmental abnormalities. The length of time for which a plant can withstand starvation varies from a few days to two months or more, and depends on the soil in which it naturally grows and the climate; but the most important factor is the ability of the plant to store food and water before the period of deprivation begins.

A number of other factors too numerous to mention, and not of especial importance in this brief discussion, enter into the effect of complete and total inanition on plant life; the mode of their action, however, is still imperfectly understood. It has been advanced as a probably tenable theory "that the essential elements in plant nutrition appear to act in two ways: (*a*) as component parts of the cell structures or fluids; and (*b*) as indirect agents in causing less understood physical or chemical conditions necessary for the proper functioning of the cell,

whether as carriers of other ions or as specific antidoting agents." (A. Reed.)

All the phenomena just discussed are the result of total inanition, or starvation. It has been stated that partial inanition, or the deficiency of one or another of the food elements or essentials, may produce vastly different effects on the body as a whole or on only one part of the structure. Of all the requirements, water is the most important. Many plants depend for their form and structure largely on the relative water content. An acute diminution of the water supply to plants causes them to wilt, while death results if they are deprived of water for too long a time. Different plants react to changes in water supply in different ways, depending largely on the character of the plant, and its natural habitat. Depending on the nature of the plant a restriction of water causes distortion of form and structure, and when water is withheld entirely, all plants will eventually die.

Calcium, potassium, magnesium, sulphur, phosphorus, and nitrogen, are all essential to plant life, as are certain organic foods; and deficiency of any one of these elements or any food, produces definite deleterious results, which are characteristic for the substance wanting in the nutriment.

It is quite likely that certain factors which are analogous to if not identical with vitamins are essential to plant growth. These may result from bacterial or fungoid growth, but act in some way as yet not understood. All vitamins are of vegetable origin, indicating a power on the part of the plant to generate them through a process of synthesis.

One factor which has always been recognized as indispensable to plant life is sunlight. The full significance of this factor in animal life has only recently been appreciated. How sunlight acts on metabolic processes is not entirely understood. In plants the effects of it may be due to "the lack of carbohydrate food normally produced through the chlorophyl. The plant is thus thrown upon its stored food material for nutrition, much like an animal during starvation." A faulty assimilation occurs, which may be due to a lack of direct effects of the sunlight itself, or to a disturbance of the water balance through diminished transpiration. Many changes in both form and structure result from partial water deprivation as well as in growth in stature. One interesting effect in some plants is that while the portion above ground may be stunted, the roots are enormously increased in size.

Turning from plant to animal life we find a striking analogy in the changes observed as a result of inanition. These are interesting and



of especial importance in the lower forms of animal life, particularly in the protozoa, since, being unicellular, they in a measure interpret to us the changes occurring under similar circumstances in man, through the cell. The period of survival after complete inanition varies with the species of the individual protozoön, from three to twenty days; the average being probably near seven days. Sudden death may occur before the usual time in cases of starvation; this as is well known also happens in man. The size is uniformly reduced and marked changes in form also occur. The endoplasm is uniformly and progressively reduced in amount, and becomes transparent, and in the later stages vacuolation is marked. In the early stages of inanition ectoplasmic structures are only slightly affected, but as the inanition progresses there are partial resorption and decrease in all vital phenomena. The nucleus is affected far less even than the ectoplasm, but may show early changes in form with loss of chromatin. It elongates and also enlarges. The micronucleus remains practically unaffected. "This persistence of the nucleus is a factor of great importance for the survival of the organism during periods of inanition." (Jackson.)

The effects of starvation on the power of reproduction of the protozoa are still in doubt; in some instance there is evidence to show that reproduction may be at least temporarily stimulated, while in others it is probably retarded. There is an analogy between food hunger and sex hunger, according to some observers. Protozoa show remarkable recuperation from inanition when refed.

As has already been stated the capacity to resist starvation varies in the several species of protozoa. But in all there are definite alterations in form and structure and in many instances also in consistency and color. The paramecia appear to have been studied more extensively than some of the other species and in them it was found that death occurred "either (1) slowly, by a process of gradual granular degeneration and ultimate disintegration, or (2) rapidly, an infrequent form due usually to a mechanical injury of the (probably weakened) cell membrane. Granular masses of chromatic (nuclear) origin are frequently long recognizable in the disintegrating dead cells."<sup>1</sup>

"Recovery of the starving paramecia was found possible by careful refeeding, if begun not later than the third to fifth day of inanition. Division recommences three to five days later. The process of recovery is exactly inverse to the process of degeneration during inanition."

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<sup>1</sup> Wallingren, *Ztschr. f. allg. Physiol.*, Jena, 1: 67-128.



When studies are made on the higher invertebrates, the problem as might be expected increases in difficulty. The various species differ to such an extent from each other that generalizations are hard to make. Such observations as have been made are instructive and throw additional light on the effect of inanition in the higher forms of life including man. In general, starvation affects these higher invertebrates in a manner quite analogous to the protozoa, but since they are more complex organisms, the process is more complicated. The endurance in the higher invertebrates varies very much more widely than in the lower forms. The length of time that starvation can be withstood depends on a number of different factors; some species die from total inanition within a few days while others endure for very much longer periods, several years having been recorded in some species. The age of the individual has a marked influence, and as might be expected the younger the invertebrate the shorter the endurance.

There is a variation observed for different individuals even of the same species. This is quite analogous to what is found among human individuals, and may be due to a difference in vital capacity. The kind of food to which the insect is accustomed, as well as the temperature to which it has been subjected have their influence, as well as a number of other environmental conditions. As in other forms of life size is affected through inanition, but is less marked, obviously, in the species having a skeleton. The reduction in size resulting from inanition is accompanied by changes in form, in some cases rendering the individual almost unrecognizable as compared with the normal. In some the structure is altered greatly, and the so-called reduction process is a reversal of the process of development.

Individual organs and tissues are affected also, but not by any means to the same extent in the same individual. Some organs are more and some less resistant to starvation, particularly desiccation, and this observation applies also to different tissues. All are affected ultimately to some extent, but while some respond quickly to starvation others resist for a considerable time. In the higher forms the skeleton is affected but slightly if at all; the growth impulse apparently continuing to function, probably at the expense of the body as whole. Only calcium deficiency appears to cause arrest of the skeleton growth, or to cause retrogressive changes.

Sexual function is practically always affected. In those species which reproduce both sexually and asexually, malnutrition favors the sexual method, the asexual being inhibited practically entirely. Mal-

nutrition acting at certain periods of ovulation, especially in the period preceding the ripening of the ovum tends to produce male offspring. Thus sex may be predetermined through inanition, probably in either direction through nutritional changes, though in some instances other environmental influences are of significance. There are conflicts of opinion, however, on this phase of the subject, and further study is needed before definite conclusions may be reached.

The effects of inanition on the body as a whole and on various organs and tissues has been discussed. These changes are of course dependent on changes in individual cells. Some cells are merely reduced in size or altered in form, while others are destroyed completely. Thus it is seen "that the decrease in the size of the body is due partly to the complete disappearance of cells and partly to an atrophy of those persisting." (Jackson.) Atrophy is brought about through a series of changes; first the cytoplasm loses its stored food, pigment and various inclusions, then a fusion into a mass having no cellular individuality takes place. Vacuolation then appears, the cytoplasm diminishes in amount and then complete absorption of some of the cells. There is a period of simple reduction of size which precedes the disintegration; some cells stop at this stage while others proceed to complete absorption. The nucleus is most resistant and is the last part of the cell to be absorbed. "Some specific changes in cell structure are noted during various forms of partial inanition. Thus calcium deficiency may loosen the intercellular attachments, but it does not affect the ciliary mechanism, and apparently permits mitosis to continue in the embryonic tissues of various invertebrates. Phosphorus and potassium, however, are evidently necessary for mitosis, as in plants. In addition to the mineral salts, certain proteins, fats or carbohydrates, water, etc., are doubtless essential to life, but we have as yet few data upon the morphological effects of their deficiencies among the higher invertebrates."

In the developmental stages of these organisms, especially embryonal, the tissues have a decidedly different reaction to inanition from the adult tissues. Certain organs not only are not affected deleteriously, but actually grow and develop; which takes place at the expense of the rest of the organism. The organism as a whole undergoes retrogression, reversing the normal processes of development.

"Underfeeding during the larval period may also result in undersized adults, and also sometimes in marked structural modifications. In some cases, these acquired characters appear hereditary, at least for

a few generations; although ultimately upon adequate diet there is an evident tendency to return to the original condition."<sup>2</sup> (Jackson.)

In some forms regeneration can take place during the period of starvation; but much absorption of older tissues occurs to supply the food for the regeneration of the younger.

Return to normal after prolonged periods of partial inanition is possible, but it is not clear at present whether there is permanent deformity, unless the inanition occurs during the larval stage. The undernourished larvæ of some species produce adult products, distinctly smaller than the normal, and this influence may extend to several succeeding generations. In some species a prolongation of the normal life span is effected through alternate fasting and refeeding.

When a study is made of the effects of inanition on vertebrates, important information is gained, which bears directly on the effects of starvation and disease on human beings. In vertebrates as in the lower forms of life, already observed, the length of time during which the organism can endure starvation varies within wide limits, and according to many different circumstances. In very small species, the duration is brief, while in others, especially in reptiles, it may extend over years. In this case the power of dormancy known as hibernation affects the results to a large extent. As in all other similar studies, we find that if all food is withheld, but water allowed, the endurance is greater. The two extremes will be found to be that large old animals endure longer than small young ones. Carnivora withstand starvation better than herbivora, while cold-blooded animals endure better than the warm-blooded. The ability to store food within the body determines the power to withstand starvation, while complete rest versus activity determines the outcome to a large extent. In other words much depends on the factors which influence metabolism generally. Vital capacity, that indefinable something, which is an individual characteristic determines not only the endurance for starvation; but in disease the outcome is often determined more by the capacity to withstand the inanition involved than to withstand the disease itself.

The relative loss of body weight is subject to many variations, and depends upon age, the size of the individual, whether the animal is relatively fat or otherwise when starvation is begun, upon species, and upon a number of environmental influences. In all animals, including

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<sup>2</sup>If such phenomena can be proven to occur in man, not only will much doubt be removed as to the influence of food on stature, but also the matter of "familial tendencies" regarding stature and even form.

man, the head suffers in loss of weight less than the rest of the body, giving the effect of an increase in size of that part of the body.

In oviparous vertebrates there is a storage of nutriment in the egg, which protects the embryo, while in mammals this protection occurs through a sacrifice of maternal nutrition. Starvation of the pregnant mother does not produce an infant with a diminished birth weight. In all animals, especially in children, growth in stature proceeds in spite of even total inanition. This growth is of course skeletal. The state of nutrition and the measure of malnutrition can be best observed through a height to weight ratio, as we have seen in the discussion on the normal and the malnourished child. "Recovery from inanition is generally possible, unless extreme stages have been reached. Recuperation under proper nutritional conditions is especially rapid in the young, but permanent stunting or dwarfing with failure to attain normal size may occur when the inanition has been severe or prolonged, and especially when occurring at a very early age."

As has been mentioned, the period of endurance of starvation varies in accordance with a number of factors. In animals there is a fairly constant period for each species. In man some remarkable instances of total abstinence from food for quite long periods have been recorded, followed by recovery. In human infants the duration of starvation, with subsequent recovery, in some of the presumably reliable records, is almost unbelievable. The best evidence at hand at present shows that the extreme limit of endurance of starvation, with water administered, is sixty days in the adult human being; in infants suffering from congenital atresia of the esophagus, death occurs in three or four days; and in duodenal atresia from four to twelve days.

Loss in weight in the adult starver varies with the length of the starving period, and depends on whether or not water has been allowed, but most important of all it varies in direct proportion to the amount of stored foods, especially fat. In some instances the loss is not so apparent, and is marked by the appearance of an edema which develops in the course of inanition, and is known as hunger edema. This phenomenon is a familiar one to those who have seen many cases of acute or subacute inanition in infancy, from disease or starvation.

All observations on starvation in animals and human beings are interesting and of vast importance as furnishing data, which when collected in sufficient quantity will enable students to clear up many existing uncertainties. It is of particular interest and importance, however, to observe what has been shown regarding inanition, particu-



larly partial inanition, in young animals, and its bearing, perhaps, only through analogy on the young human being.

During inanition in the young there may be an actual loss of weight, or a failure to gain in weight. Or there may be retardation in normal growth rate. It must not be forgotten that in a *growing* animal failure to gain constitutes an actual loss. In the adult, where growth has ceased, a stationary weight is of no significance, and in fact is normal so long as the body functions are maintained. But this is not the case in the young. Resistance to inanition in the young is slight, but increases progressively as maturity is approached. The needs in the young are relatively greater, because of the growth factor; and as a matter of fact a growing organism may starve to death on a diet sufficient to maintain the body without a loss of weight.

The loss of weight is determined by the age of the individual and the amount of food deprivation. Death from starvation is determined in large measure also by the age of the individual and by the percentage of the body weight lost. These factors vary in their influence widely according to species, and also individually. "A diet below the maintenance requirement will produce death more rapidly with actual loss of body weight, while a diet above the maintenance requirement, but still subnormal in amount, will cause a retardation in growth proportional to the degree of deficiency." (Jackson.) This is one of the most important observations yet made, since it bears directly on the problem of the undernourished growing human organism.

We have already seen that in mammals, starvation on the part of the pregnant mother affects very slightly if at all the birth weight of the offspring. On the other hand the younger the individual the more susceptible it is to starvation. It would follow from this last statement that the organism is unusually susceptible during the embryonic stage. Nutrition is maintained at the expense of the mother. Jordan has observed (already alluded to) that some embryos are found which are abnormally small for the period of gestation. Such observations as this might justify the assumption that at a very early stage of embryonal life impaired nutrition may affect the size of the organism. Whether there is a recuperation, through excessive nutrition, later in fetal life, whereby the organism is enabled to catch up, or whether such embryos remain small, cannot be determined at present.

Observations made on animals by different students have shown vastly different results, in many instances quite contradictory, so at present final judgment must be suspended. So far no definite evi-



dence exists that size and development in the human being are influenced by prenatal environmental (nutritional) influences.

That for the first few days of life, in practically all animals, there is a loss of weight, is a familiar phenomenon, certainly in the case of the human infant. This phenomenon has already been discussed in previous parts of this volume. Such loss is usually thought of as being physiological, and this is in part no doubt correct. It is due in some measure to the emptying of the bowels of the contents, present at birth—meconium; loss from urinary excretion; loss of fluid from the skin; and moisture and carbon dioxid from the lungs. Some of this loss is due to the body discarding the waste products of metabolism, which continue as vital processes, regardless of the fact that no food is ingested to supply what is consumed. When no food is furnished the newly born infant, a state of inanition is produced; but certain symptoms often appear during the first few days of life, even when an adequate supply of food has been given. The theory has been advanced to account for this phenomenon to the effect that the digestive system does not adapt itself to the sudden work put upon it. This theory seems scarcely tenable; for there is rarely any evidence of indigestion in the newly born when food is first taken; and too, a number of experiments have been made in which food administered immediately after birth gave no evidence of digestive disturbance. It is also true that the initial loss occurs *before* any food is taken.

That a considerable amount of this initial loss is due to water insufficiency can hardly be doubted. The return to the birth weight occurs on an average of twelve to fourteen days. What harm if any results from this initial loss, is not known. The growth continues in spite of the loss of weight. That this loss can be prevented partially or even entirely has been proven by several series of experiments. That return to birth weight may be shortened by from four to six days, even when no effort has been made to prevent the initial loss, has been proven by observations on the newly born infants in the University of Virginia Hospital. Nothing definite can be said in favor of or against such efforts, since too few observations have been made.

The immediate effects of complete starvation in infants and young children have been observed grossly in cases of acute inanition, where death occurs promptly in many cases, with relatively slight loss of weight. It is entirely likely that in such instances water deficiency plays an important part; the thickening of the blood and concentration of protein, largely secondary to the water inanition, are contributing

factors in the final results. Inanition, partial or complete, leads to the condition known as infantile atrophy, or marasmus. This condition has been studied clinically in a previous section. It hardly justifies classification as a clinical entity, since it usually occurs in the course of or following other illness, or some disease which produces the starvation. Although it is often due only to insufficient food, either quantitatively or qualitatively, and presents definite symptoms, which in large measure are those of starvation, it is well to include it in textbook classifications. In some instances not well understood, and as yet not capable of identification, the cells themselves appear to be at fault, in that they are unable to assimilate food regardless of the amount carried to them, although it may be apparently normal in both quantity and quality. This has been said to be due to intrinsic protoplasmic defect. Intrinsic defects of the cell which act in this manner are said to be congenital and in some instances even hereditary; but in most cases are probably acquired, perhaps often through the action of toxic material generated somewhere in the body. The greater part of the discussion on the cause of cell deficiency is entirely speculative; the fact is that nothing definite is known about it. There can be little doubt that in infections of the intestinal tract, the secretion of digestive enzymes is interfered with; in this way inanition may be easily produced, though ample food is ingested.

Some observations have been made to ascertain to what extent loss of weight can take place without causing death. It is difficult to make definite statements regarding this, since some infants suffering from acute inanition die without much loss of weight, while others survive after an enormous loss has occurred. The personal element, whatever that may be, enters to a marked degree, as well as a large number of other factors, whose influence can be judged only theoretically, and yet are recognized in the individual patient. The "state of vital forces" probably means as much as any other term. Under the influence of either underfeeding or improper feeding, usually called unbalanced nutrition, the weight may remain stationary, but this as we have seen means actual loss in growing children, or there may be actual loss or decline in weight. The results in such cases depend on the duration of inanition and the degree of malnutrition attained. When the malnutrition has not proceeded too far, recovery under adequate feeding is prompt. It has long been noticed that artificially fed infants are usually of lighter weight for their height than those fed with human milk; the growth also may not be quite up to normal. This was undoubtedly

true under the older methods of artificial feeding, but the more recent methods, with concentrated food (such as acidified milk) as outlined in the section on Feeding the Normal Child, apparently develop children who are quite up to the average breast-fed ones, in both growth and nutrition. A low weight for height ratio generally signifies undernutrition, but cannot be accepted absolutely as a criterion without other evidences, largely clinical, except where the low ratio is extreme. This phase of the subject has been fully discussed in a former section.

As has already been observed, certain more or less definite deformities occur in the growing organism when subjected to inanition. These are probably brought about by a continuation of growth in some parts, apparently at the expense of other parts. This may be due to a sort of sacrifice on the part of the organs showing retarded growth, giving, as it were themselves, that another part or organ might grow and develop; or it may be that the inanition affects one part severely, due to the type of reaction of the cells of that part, while another part is only slightly affected and hence continues to grow, though improperly nourished. In either event the result is deformity of the body with altered contour, or form.

There is uniformly an increase in length; a loss of weight first in the fat deposits and next in the musculature. The head increases in weight, probably also the brain; this, together with the loss of weight of the rest of the body, and the continued growth of the skeleton, causes a noticeable disproportion between the head and the body. This disproportion is more apparent than real. There are more marked differences in other organs. Animal experiments have shown that underfeeding of a pregnant mother during the latter part of pregnancy has produced offspring which were disproportionate; the head and limbs being disproportionately heavier than the trunk. We have already seen that the birth weight of human newborns is not affected by starvation in the pregnant mother, but there are no data available showing the relation in length between head and limbs and trunk. The disproportion depends on the developmental period at which the fetus is subjected to inanition.

An abnormal growth in length of extremities occurring during febrile conditions has been described and has been attributed to the stimulation of the epiphyseal cartilages by certain toxins. Whether such a phenomenon actually occurs or is only apparent is not certain at this time. It has been claimed that acute infections of short duration affect weight, but have no influence on growth (height); on the

other hand malnutrition due to prolonged infection causes cessation in the increase of weight and also retards stature growth. It is likely that inanition occurring in young infants affects increase in height little if at all (growth impulse is strong at this time) while inanition occurring later may retard increase in height. All states of inanition if sufficiently prolonged may produce apparently permanent changes in the proportion of adult measurements. Children who have been subjected to extreme inanition for prolonged periods show retardation of growth; or at least many children who are very much under the average height for their age, give a history of having suffered from prolonged malnutrition. Underweight for long periods following malnutrition is quite familiar. "These children may yet become normal, but it is quite possible that permanent dwarfing may occur as a result of severe or protracted inanition during infancy or childhood, in accordance with the results obtained by experimental inanition upon animals." (Jackson.)

We have been considering the effects of inanition on the body due largely to quantitative deficiency of food; which might be regarded as a deficiency in calories. This has been studied from the point of view of complete or partial starvation, without special attention to a deficiency of one or another food element, though the effect of the lack of a single food element has been mentioned more than once. Food deficiency is also to be considered from the standpoint of deficiency of the individual essential food factors, protein, fats, carbohydrates, inorganic salts, vitamins, and water. Water has been largely studied in partial food deficiency in general. In studying the effect of food deficiency on growth it is often difficult to tell exactly to what these effects are due, that is, whether all of the symptoms are due entirely to caloric deficiency, or also to elemental deficiency. In any prolonged quantitative restriction of food, there occurs at the same time a deficiency in certain essential factors, the symptoms due to each restriction often overlapping. Manifestations of deficiency of any kind depend further on several other conditions: "(1) The amount of each substance which is stored in the body as available reserve; (2) the rapidity with which each substance is consumed in the body (which in turn depends upon various factors influencing metabolism); and (3) the intake of each substance in the food, in case of incomplete inanition. The exact character of the deficiency in total inanition, either complete or incomplete, is therefore exceedingly variable according to the species, age, individual, environment, etc." (Jackson.) In infectious proc-



esses, it is further difficult to determine to what extent certain manifestations are due directly to inanition, or to the absorption of toxic substances generated as a result of the disease, or even to faulty metabolism accompanying the disease. In the handling of infectious diseases accompanied by considerable wasting, it is hard to draw definite conclusions, as to the cause of the symptoms. It is difficult to handle the disease from the standpoint of nutrition. Just how far we should try to combat the wasting by a diet approximating a maintenance ration, and yet remaining within the impaired digestive powers of the individual, is problematical. Such a problem is present in an interesting fashion in the case of typhoid fever. When this disease was treated with a much restricted diet, amounting to partial starvation, and in many instances to extreme starvation; not only was the individual emaciated at the end of the active condition, but the return to normal weight was usually slow. In some cases however the patient gained so much weight during convalescence as to far exceed the weight before the onset of the illness. Excess of weight increase after starvation diet has been abandoned is not unusual. Delirium is general in typhoid fever and has been supposed to be due to the infection or the febrile condition. Since a high caloric diet has been the rule in the handling of this disease, delirium is less marked, and the loss of weight is not only far less than on the restricted diet, but in many instances the patient can be made to gain while suffering from the disease. To what extent the digestive powers are impaired by this disease we do not know; nor do we know whether the high caloric diet imposes an unwarranted strain on them, but from personal observation on a fairly large number of cases handled in this way, no apparent harm has resulted. This is mentioned merely to show how difficult it is to steer between the Scylla of inanition and the Charybdis of the infectious process, since it is well known that when nutrition is maintained infectious processes are overcome with greater ease and less impairment of body waste. The maintenance of water balance is also of prime importance and probably alleviates as many of the symptoms as does the food.

It is now well known that protein is the most important of the food elements to the growing organism. Since there are a number of different proteins, furnishing varying amounts of amino-acids; and since the amino-acids differ in their effect on the organism, due consideration must be given, not only to the amount of protein, but also to which of the amino-acids the particular protein furnishes. In the growing organism animal protein is of far greater benefit than that of vegetable

origin. It is difficult to determine exactly to what extent the various deficiencies affect the growing organism; but through the study of certain deficiency diseases we are enabled to evaluate the various food elements in relation to vital processes, by observing their effect in producing certain symptom-complexes; the effect of their administration in curing these diseases when once developed, and finally in preventing their recurrence or their occurrence.

The effects of the most important of the proteins have been studied in this volume, in the course of our discussions of the various deficiency diseases and of other conditions of impaired nutrition; and also in the descriptions of the food elements themselves and their relation to bodily function.

It is pertinent, however, to ask what appears to be at this time an unanswerable question: In the arrest of growth (stature) which is the more important factor, the deficiency of total calories, or the deficiency of growth promoting vitamins? Theoretically, the absence from the diet of these vitamins will cause a cessation of growth, regardless of the number of calories taken, the latter having to do largely with weight increases. This theory has been advanced by some very excellent observers. It would seem, however, that vitamins cannot promote growth, unless sufficient calories are also furnished, since growth is a part of the general body processes. It must be recalled, however, that growth continues during inanition, even though the body itself is wasting. That weight and even stature can be controlled at will by the administration of definite amounts of food of definite qualitative value has been proven by experiments on lower animals by several observers, notably by the recent work of Mendel and Osborne; as a result of this it is strongly implied if not positively stated that the same thing can be done in the human being, even to the extent of producing oversized adults (bordering on gigantism) through feeding by direction during the growing period.

In some instances two essential food factors are contained in the same food sources and therefore it is difficult to say to which of the elements the peculiar symptoms are due. Likewise the retention by the body of one element is dependent on the ingestion of another element and so the richness of food in essential elements plays an important part in the maintenance of a balance. A deficiency of water causes failure on the part of the body to retain certain salts; and this interrelation is also reversed, the retention of certain salts influences to what extent water is retained. It must be also remembered that a subacute or latent

stage of all deficiency diseases preceded the appearance of specific manifestations; and during this stage there is a general state of malnutrition (though not necessarily undernutrition), as shown by loss of weight, which ultimately culminates in a specific disease entity with characteristic symptoms and manifestations.

Water inanition has already been alluded to and formed a part of the discussion on partial and complete inanition; it was discussed briefly under anhydremia in the description of malnutrition. That water is essential to life and growth is well known. When the body is deprived of water beyond a certain limit various symptoms appear, specifically the febrile reaction in the newborn as shown in the condition known as "inanition fever," more recently termed "dehydration fever." That water deficiency alone can cause sharp elevation of temperature in the very young there can be no doubt; it is entirely likely that concentration of body tissues, especially the blood, which accompanies this condition, and the relative excess of protein, without proper elimination, play parts in this picture. That food has not been ingested in adequate amounts at the time of this reaction, thus causing a metabolic imbalance, no doubt has an influence. That the presence of water in sufficient amounts, or the deprivation of body tissues of sufficient amounts of water below what is called the fluid balance of the tissues, affects the living organism to a marked degree in one or the other direction, is a certainty beyond question or doubt. A large portion of the weight of the body is water, and gain or loss in weight is determined more by the amount of water retained in the tissues than by any other single factor. In young infants as little as one half ounce of water more or less than the normal requirement in twenty-four hours will decide whether the infant gains or loses.

Each tissue and each organ of the body is affected by inanition in some way. Each tissue and each organ has a specific reaction to deprivation of one or another of the various food elements. Each essential food element has a specific bearing on the nutrition of at least one tissue or one organ. It is not feasible to attempt a discussion covering all of these matters in a volume of this kind; reference being made to the studies of deficiency diseases and to more extensive works which deal directly with the specific problem involved.

One of the most interesting phases of the whole question of the effects of inanition on the body is the actual cause of death in either total or partial starvation or inanition. A very large number of theories have been advanced to account for the fatal outcome. Only a

few will be mentioned. One of the earliest of these theories is that death is due to exhaustion of stored food materials.

Another is that asphyxia causes death, through absorption of accumulated toxic material, causing paralysis of the respiratory center. Still another theory suggests that death is produced by auto-intoxication, from toxic substances resulting from impaired metabolism. It is well known that impaired nutrition lowers the vital forces of the body and makes them less resistant to infection. That a number of individuals who are the victims of inanition from one cause or another die of infectious processes, particularly bronchopneumonia, is well known. In the depleted condition of the body resulting from starvation, an infectious process may cause death without any apparent reaction on the part of the body; the infection existing without being recognized. The actual cause of death from starvation therefore varies with circumstances and with the individual, and for the moment at least no general statement can be made.

The whole question of malnutrition and its immediate effects and ultimate results is one of the most far-reaching and important in modern life. It is of especial interest to those who are striving for a solution of the vast problem of making a better generation, at least physically, of adults out of the material presented to us in the present generation of growing children. Long-continued inanition as we have seen affects all tissues of the body; one tissue, however, calls for special notice, and that is muscle. As a result of inanition, complete for a short time, or incomplete over a longer period, the muscles atrophy to a considerable extent. If such atrophy with its consequent impairment of muscle power occurs at a time when the activity of the muscles is great and the child is growing and adapting itself to life's burdens, serious consequences result. During the periods known as the pre-school, the school age, and youth, the body is taking on its permanent form, posture is being fixed, and muscle power is developing. During the period of youth the adult is arriving and a definite amount of muscle volume and muscle power is needed to hold the skeleton in the normal posture. Unless defects are corrected by certain exercises and definite training, and unless the child is made to increase in weight, a faulty posture in the adult is inevitable. This is especially apt to occur where the child is made to do manual labor during these years. Unfortunately relatively few children are given sufficient care during these formative years to prevent such a misfortune or to correct the defects when they have developed. The results of such a condition extend well into adult



life. Faulty posture lowers the capacity of the individual for many forms of activity. This was forcibly brought to our attention, as has been mentioned, during the World War when many drafted men were found to be incapable of enduring the hardships incident to warfare. Not until physical trainers had worked on these men, correcting their faulty posture, could they serve their country adequately. Women with faulty posture are often subject to uterine displacements, which make for difficult menstruation and difficult labor, and render them unfit for household duties and the very arduous and important duties incident to the rearing of a family. The wage earner is not capable of doing his best when handicapped by a faulty posture. Manual labor of all kinds calls for well developed muscles; while those doing mental work are seriously handicapped if physical endurance is impaired. But above all, the business of being a parent calls for all that is in an individual, both physically and mentally; and since it is not at all unlikely that undernutrition with consequent bodily defects may be transmitted to several succeeding generations, it behooves all who are in any way responsible for the welfare of children to aid in the prevention and correction of inanition-malnutrition, and thus raise for the benefit of mankind a stable citizenry, of enduring and efficient capabilities. The problem of inanition, or malnutrition, by whatever name one may choose to designate it, is therefore not alone a medical problem but an economic and political one as well. This problem cannot be attacked and solved by ministering only to those children who are brought to the attention of a physician, because they "are not up to standard," but can only be handled in any far-reaching manner through the education of all the people as to the significance of the problem, and by enlisting the support of all those in any way in authority over children.

## CHAPTER XII

### NUTRITIONAL DISEASES

#### SCURVY

Scurvy occupies an interesting and unique place in the list of deficiency diseases. It has not only been recognized for a long time, but it was also known that it was due to "something lacking in the diet" and that by supplying that something the disease could be cured and even prevented. It was found that the juice of oranges and other citrous fruits would both prevent and cure the disease, and travelers among primitive people have told of the administration of certain vegetable juices to infants, apparently with the idea of preventing a condition which from their descriptions was in all probability scurvy. It was not until Eijkman in 1897 demonstrated that the polyneuritis of fowls was due to a definite food deficiency, and a certain analogy was seen to exist between polyneuritis and other diseases now recognized as deficiency diseases, that attention was directed to scurvy as belonging in this class. These observations stimulated the investigations of Holst and Fröhlich which definitely proved the truth of the theory.

For a long time the symptoms presented by scurvy were discussed by a number of observers, and attributed to several other diseases now recognized as separate and distinct conditions. By the earlier writers, and in certain foreign countries by some writers of to-day, scurvy and rickets are always considered as variations of the same condition, scurvy being thought to be the acute form of rickets. Although some earlier writers recognized that the disease in infants now known as scurvy was at least clinically identical with adult scurvy, it was Barlow who first identified through anatomical proof the condition as the same.

Although prevalent in most countries, it varies in frequency, judging from the literature, to such an extent that either it is not recognized, which is unlikely, or methods of feeding in these different countries must account for the difference in incidence. Scurvy has always been a serious menace to troops in time of war, to the civilian population in time of siege and famine, to mariners in the days of the sailing vessel

and the slow steamer; and to infants who are artificially fed. As a result of our newer knowledge of cause and cure the incidence of this disease has been very much reduced.

**Etiology.**—So long as the specific etiology of a disease remains unknown, numerous theories are proposed to account for its symptomatology and pathology. The case of scurvy proved no exception to this rule. Among the theories proposed were: the lack of mineral salts, particularly calcium; the lack of citric acid; food intoxication; infections, both enteral and parenteral; and others which call for no mention. In adults it is quite likely that infections play a part in precipitating the onset of scorbutic manifestations when the vitamin deficiency is not great enough to produce symptoms otherwise. In infants infections no doubt precipitate acute symptoms, as in adults; they determine the outcome in many cases. The specific cause of scurvy is now recognized as a deficiency of the antiscorbutic vitamin or water-soluble C.

Vitamin C is contained in large quantities in the juice of citrous fruits but most abundantly in the juice of the orange. In this form also it is most conveniently added to the daily diet, when called for to augment a known or suspected deficiency. It is found also, but to less extent, in the juice of the tomato, and still less in a number of the green vegetables, notably those of the leafy variety or the leafy portion of others (see Vitamin Content of Foods, p. 283). It is abundant in human milk and to a less extent in the milk of the cow. This vitamin is the least stable of the known vitamins, being especially affected by heat and the process of oxidation. Any influence also which causes drying of foods containing the vitamin, is especially detrimental, and in most instances destroys it. The above statements, however, should be modified somewhat in order to conform to facts. Prolonged heating affects the vitamin, often vitiating its antiscorbutic action entirely, while higher temperatures, if of short duration, impair its efficiency only slightly. Thus pasteurization of milk destroys a large portion of the vitamin content while boiling affects it but little. The fact that the vitamin is sensitive to oxidation may account in a measure for its destruction during pasteurization, since this process calls for heating for a relatively long time, and is not always performed in tight containers; hence oxidation occurs at the same time, and both influences act together. Where the pasteurization is performed in approximately airtight receptacles oxidation does not occur. The same is true of superheating. In boiling, the heating process is short and oxidation does

not take place in this brief time, at least to an extent sufficient to impair the vitamin activity. Dry, powdered preparations of milk are also prepared in relatively air-tight containers, and besides, the process is short; hence the vitamin content is only slightly affected.

Solution in an acid medium protects the antiscorbutic vitamin, as shown by the lasting potency of orange or tomato juice. Prolonged boiling of any food containing this vitamin destroys the vitamin, and so, many articles of food ordinarily valuable for their vitamin content are rendered worthless by certain methods of cooking. On the other hand modern processes of canning conserve the vitamin in spite of the high temperatures to which the food is subjected, because of the sealed or vacuum method, thus diminishing oxidation.

How the antiscorbutic vitamin acts in preventing scurvy is not known. Its prophylactic and curative qualities depend on the quantity of the vitamin taken into the body. A relatively large amount is needed for either purpose and the minimum amount is fairly well known. It is not known whether all of the ingested vitamin reaches the cells, or whether a part or any of it is digested by the stomach or intestine. How much if any is stored by the body is still in doubt, but it is fairly certain that whatever is stored, remains only for a very short time. Its action, certainly during the curative process, appears to be quite prompt.

So far it is reasonably certain that scurvy is a condition which affects almost exclusively infants who are artificially fed. Under exceptional circumstances where mother's milk is scant, and the food of the mother is sufficiently poor in vitamin content, nursing infants may be affected; a few examples have been reported in this country; a relatively large number have recently been reported abroad. Many of the reported cases are open to much doubt.

As has already been intimated, scurvy is induced in infants fed for long periods on pasteurized milk; but even this depends on the method used in the process. Sweetened condensed milk is said by Hess not to cause scurvy, though he admits that this may depend on the vitamin content of the food of the cows from which the milk was derived. I have repeatedly seen scurvy develop in infants fed exclusively on sweetened condensed milk.

Some proprietary foods are the greatest offenders in the production of scurvy; that is to say, in powdered form and mixed with water or an insufficient amount of fresh milk. In my own experience the largest number of cases developing this disease have been fed on some form of malted milk. Infants fed on fresh cow's milk rarely, if ever, develop



the affection. The food of the cows producing the milk determines the amount of milk required to prevent the disease. It has been stated that one pint of fresh cow's milk per day will prevent scurvy in an infant, provided the milk is rich in vitamin content. Where the milk is relatively poor in vitamin a quart per day will be required. Prolonged and frequent handling of milk before use by the infant appears to bear a direct relation to the incidence of the disease.

The scurvy of infancy is distinctly a disease of the second half of the first year of life. From six to nine months are required for the disease to develop, from its incipency to the florid stage. Unlike rickets, which is largely, and marasmus, which is almost entirely, a disease of the poorer classes, scurvy affects the rich and poor alike. In my own experience the poor have been affected more frequently than the well to do, though the latter are often prone to make use of high priced and much advertised infant foods, which are in most cases poor substitutes for raw, or freshly boiled, cow's milk.

**Pathology.**—The most striking feature of gross pathology is the presence of subperiosteal hemorrhages. The most frequent sites are the periosteum of the femur, particularly the lower end, though the whole shaft may be affected, and the upper end of the tibia. Changes occur at the costochondral junction and produce a beading closely resembling the rosary of rickets. There is an infiltration of the soft tissues which are anasaruous on section. The muscles are also infiltrated and pale in color. Serous cavities such as the pericardium, pleura, peritoneum and joints contain fluid in varying amounts. There is definite and almost constant enlargement of the heart. The lungs are congested, as is the intestinal tract, particularly in the upper portion. Ulcers may be found, probably due to secondary infection. The liver and spleen are not affected.

The most prominent microscopical changes occur in the bones. The characteristic changes are found at the junction of the diaphysis and the cartilage, the typical change being best demonstrated at the costochondral union of the ribs. "Under the microscope the line of juncture is not sharp and straight as it is normally, but presents a wavy or irregular contour, the cartilage jutting into the bony end of the rib, instead of abutting in neat apposition to it." (Hess.) There is in addition much distortion in the arrangement of cells and signs of recent hemorrhage. There are few osteoblasts, and occasional spindle and star-shaped connective-tissue cells. The cartilage is also affected; the proliferating columns in the central convex portion disappear, and this

presents one of the early signs. The osteoblasts are not functioning normally; the osteoclasts are not in excess and "the resulting lesion is clearly the result of almost normal bone resorption with a lack of bone regeneration." (Hess.) Hemorrhages occur in the bone, lungs, muscles, and occasionally in the liver, spleen and kidneys. There is a definite but undetermined change in the vessel walls. In fatal cases there are focal degenerations in the lumbar cord, and the peripheral nerves are frequently the site of hemorrhage.

In experimental scurvy it has been shown that the various tissues entering into the formation of the teeth are seriously affected. There are softening of the dentine and irregular arrangement of certain groups of cells. Even though the degenerative process has advanced to a point which would make repair seem quite unlikely, the administration of orange juice causes prompt regeneration of tissues and hardening of the dentine. A definite change may be noted in twenty-four hours. In the light of these findings it is quite likely that scurvy may exert a definite influence on the time of eruption of the teeth as well as their condition after eruption.

**Symptomatology.**—It is likely that in all deficiency diseases there is a period of faulty metabolism, or malnutrition, during which certain changes occur without producing signs or symptoms characteristic of the disease; during such periods the patient may or may not lose weight, or the weight may remain stationary, which, in the case of children, is equivalent to actual loss. There may be indefinite symptoms, such as not being quite up to the usual standard, but no symptoms or signs appear which might lead to a diagnosis. Not until the condition progresses to an advanced stage of faulty metabolism do signs or symptoms develop which are recognized as the classical and typical ones of a condition to which we give a name. This sequence is well recognized in scurvy. According to the duration and intensity of symptoms there are three types or stages: latent, subacute, and acute or florid.

#### LATENT SCURVY

The latent type corresponds to the period during which florid scurvy is developing, and requires about six months of partial or total vitamin deficiency. This might well be called the prescorbutic stage. There are no definite manifestations and it is with difficulty that the condition is recognized. The child affected begins to show a lack of desire for food, or turns against a favorite article of food. The

weight is either stationary or a definite loss occurs. This loss is usually slight, or the child may show slight gain one week, while the next there is either no gain or a slight loss. The child becomes fretful and its disposition may undergo a marked change in a few days or weeks from happiness to a whining discontent. There is an apparent anemia, the complexion being pale or sallow. There is not only restlessness but the reflexes, particularly the patellar, are exaggerated. The pulse is rapid as is often the respiration. There are no hemorrhages at this stage, though it is stated that there may be a bluish red line in the gums just above the teeth. Whenever such symptoms as these develop a careful history of the diet may disclose the cause, but in many instances it will not aid in a diagnosis, since the child may be on milk relatively poor in vitamin, and this is difficult to ascertain without lengthy laboratory methods. The therapeutic test usually clears the matter promptly.<sup>1</sup>

#### SUBACUTE SCURVY

The subacute form of scurvy is more severe than the latent form and far less severe than the acute or florid type. The signs and symptoms are those of the latent form, somewhat exaggerated, to which are added certain additional manifestations. Whereas the latent form may show signs prior to the sixth month, because of the fact that it is in the advancing stage, the subacute form usually appears after the sixth month. The weight curve closely resembles that of the latent form, in that there is apparently slight or no striking state of malnutrition. This is in part due to the retention of fluid in the tissues, since there is always some degree of infiltration, and evident edema is common. This may be confined to the eyelids, or it may show in the lower extremities. A striking characteristic of this edema is that it does not pit. The signs and symptoms referable to the nervous system are those of the former type, but may be slightly more pronounced. Restlessness and fretfulness are the rule, while anorexia may be pronounced. There is often a slight, continuous or intermittent tenderness of the legs. The nurse or mother may observe that the child sometimes acts when handled as if in pain. The respiration and pulse are accelerated; the urine is diminished and is usually normal, but may contain a trace of albumin, and an occasional blood cell of each variety. There is almost always present some change in the appearance of the gum margin. There is a bluish red line or an actual slight hemorrhage at the same

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<sup>1</sup> Hess, *Scurvy Past and Present*, Lippincott.

point, but this is rarely constant and may appear and disappear from time to time. Very occasionally in this type there are a few scattered petechial spots. After a prolonged attack of coughing there may appear a subconjunctival hemorrhage. This type is the one most frequently seen and should be easily recognized. Prompt treatment effects a rapid alleviation of symptoms.

#### ACUTE OR FLORID SCURVY

The acute type is the one to which the classical description of scurvy has been given. The picture is a pitiable one. The child is at least six months of age. It lies motionless and quiet except when disturbed, or when approached. The slightest movement of the bed will cause it to cry out with pain. Many authors describe such cases as undernourished. In my own experience, although I have seen several thin infants with scurvy, the majority have had at least the superficial appearance of being well nourished, and most of them appeared fat, but some of these cases are edematous. Of course malnutrition is present, but not always undernutrition. The posture is quite characteristic. The child lies on its back with the thighs flexed and everted. One or both thighs, and one or both legs, are swollen and exquisitely tender to the gentlest touch. This often leads to a diagnosis of rheumatism, and this indeed is the diagnosis in most cases sent to a consultant. Examination of the mouth discloses gums spongy, bluish red in color and which bleed on the slightest touch; in many instances the bleeding occurs spontaneously. The urine is scanty and often contains free blood. Albumin and casts are the rule. In some cases bloody urine is the first sign to attract attention. Ecchymotic areas are scattered over a greater part of the body. The eyeball is occasionally bulging, due to orbital hemorrhage. There is generalized edema, the face being affected to a marked extent.

The outstanding pathology of scurvy is, as we have seen, hemorrhage. Subperiosteal hemorrhage is the characteristic sign of the condition. The lower end of the femur is most frequently and characteristically affected; next in frequency the upper end of the tibia; and rarely the bones of the upper extremity. The joint itself is rarely affected, although these hemorrhages are near it; the swelling incident to the hemorrhage may develop suddenly, but more frequently its development is gradual. This differentiates scurvy from rheumatism. The hemorrhage may be sufficient to cause only slight enlargement or



so great that the thigh or leg appears twice the normal size. Although the x-ray shows the hemorrhage plainly, operations are not infrequently performed for a mistaken osteomyelitis. Under treatment the hemorrhagic condition of the gums subsides rapidly, the subperiosteal hemorrhages undergo slow absorption, remaining for long periods after the acute symptoms have subsided. The periosteum becomes calcified in some instances, in such cases a distinct crepitation may be felt. There has been some debate as to whether the hemorrhage is subperiosteal or originating at the diaphyseal-cartilaginous junction. It occurs in both places, but is probably more profuse in the latter place, and from there dissects up the periosteum. As a result of the bleeding there is frequently separation at the junction alluded to, and in some extreme cases the hemorrhage may be so great that the whole periosteum may be dissected from the shaft of the femur, which may float in a sac of blood. Complete recovery occurs after treatment, and no impairment of function remains.

There is a beading at the costochondral junction similar to but not identical with the rosary of rickets. The scorbutic beading is sharp and angular, while that of rickets is rounded.

Enlargement of the heart is now considered a constant accompaniment of scurvy, and can be elicited by percussion as well as by means of the x-ray. This enlargement, although no pathology is found on microscopic examination, is of definite significance, since the pulse, ordinarily rapid in scurvy, is perceptibly accelerated on even slight exertion.

Rarely there is found what appears to be a true nephritis which subsides as improvement in the general condition of the patient occurs under rational treatment. This of course opens the question whether this is a true anatomical nephritis or a local manifestation of a generalized dyscrasia of metabolic origin.

That the hemorrhagic tendency in this disease is not due to prolonged clotting time has been amply demonstrated. A permeability of the vessels is present. There is usually a secondary anemia, which is frequently atypical, in that the hemoglobin is uniformly reduced, whereas in many cases there is no reduction in the number of red cells. The leukocytes are normal except in the presence of intercurrent infection.

Some cases of scurvy show slight elevation of temperature. This is probably not due to the scurvy but to infections, which are common in this condition.

There is not only a metabolic disturbance resulting in varying degrees of underweight, the amount of edema often masking this manifestation, but there is actually cessation of growth due to avitaminosis.

**Diagnosis.**—The diagnosis in the florid type presents no difficulty, and it should be recognized almost at a glance. In spite of the distinctiveness of the clinical picture, errors continue to be made all too frequently. When cases of the subacute and latent type are encountered, it is safe to say that the vast majority of diagnoses are missed. If careful histories of the previous food are taken in all cases of infants coming under the care of a physician, the condition should be suspected when the symptoms outlined previously are present and the food has not been well balanced and probably lacking in vitamin.

From rheumatism it should be diagnosed by the location of the swelling which is in the joint in rheumatism and near the joint in scurvy, and by the age of the patient, scurvy appearing under the twelfth month, while rheumatism is rarely seen in infancy. From purpura the diagnosis may appear difficult at first since the gums are frequently affected in both conditions, and subcutaneous hemorrhages are common to both. In purpura hæmorrhagica, the form for which scurvy is most likely to be mistaken, the blood platelets are always greatly reduced, in typical cases to as low as 40,000 or less, while in scurvy they are increased above 300,000. Many infectious processes are accompanied by subcutaneous hemorrhages but are not apt to be mistaken for scurvy. Epiphysitis sometimes causes difficulty in diagnosis; the leukocyte count should make the differentiation. The enlargement of the bone, especially when the blood has clotted and has become hard, has been mistaken for malignant disease of the bone. The history of the case and of the feeding should prevent this error. Syphilitic pseudoparalysis has been confused with scurvy, but here also error may be avoided by recalling the fact that the syphilitic condition occurs much earlier than the scorbutic, and the blood test will usually clear up any further doubt. Scurvy has been mistaken for a large number of other conditions, but the differentiation is so manifest as to call for no particular notice.

**Prognosis.**—When antiscorbutic diet is administered promptly, recovery is certain so far as the disease itself is concerned. Reports from central Europe indicate that death from scurvy is by no means infrequent since the World War, owing to the scarcity of antiscorbutic remedies and indeed of food itself. Death occurs in adults and less frequently in infants from heart failure due to the weakening effect of

the disease on the heart muscle. When we recall that subacute and latent scurvy frequently pass unrecognized and since it is certain that the lowered vital capacity incident to scurvy predisposes to infections, the part played by this disease in infant mortality is hard to estimate.

**Treatment.**—By far the most important consideration in the treatment of this disease is prophylaxis. Nursing infants do not develop the disease as a rule. The rare exceptions are due to a very scant supply of milk from the mother or to the food of the mother being exceedingly poor in vitamin content. Of course both factors may be combined. Therefore an infant nursed for the first six months of its life is practically safe from scurvy. Likewise a sufficient amount of fresh cow's milk will prevent its development. Whenever pasteurized milk or even milk which has been boiled while still fresh is used it is far wiser to begin the administration of orange juice, or, where this is not available, the juice of the tomato, as soon as the substitute for mother's milk is begun. All proprietary foods not mixed with a sufficient amount of fresh cow's milk should be avoided. When administering orange or tomato juice, one teaspoonful of the former or two of the latter should be given daily to an infant of one month and this should be increased until at the age of three months it is receiving one tablespoonful of orange juice or two tablespoonfuls of tomato juice each day. The tomato juice should be strained and one should bear in mind that it contains just about one-half the amount of antiscorbutic vitamin as the juice of the orange. A number of vegetables are relatively rich in the vitamin, and may be used when orange or tomato is not available, though their juice must be used in much larger quantities than that of either orange or tomato. In southern states it was customary in former years among Negroes and the poorer whites to give pot liquor at an early age. This was the liquid in which a number of vegetables had been boiled. Usually a thick slice of bacon was boiled at the same time, to give flavor. This was in effect a good essence of vitamins and no doubt prevented scurvy to no inconsiderable extent.

The effect of antiscorbutics on the clinical picture represents the most dramatic example of specificity known to medicine. To witness the change from a condition of abject misery to one of contentment and happiness within two or three days is startling. Orange juice should be administered as soon as the condition is diagnosed, 2 ounces per day being a good average dose. Tomato juice should be given in twice this quantity. In addition to the specific therapy the food should be checked up at once and changes made in accordance with the errors

found. Where pasteurized or boiled milk has been given an abrupt change to unheated milk should be made. Where the quantity of milk has been insufficient it should be increased to meet the requirements of the patient. If proprietary foods have been used they should be discontinued at once and fresh milk substituted.

As has been stated, the amelioration of symptoms is prompt, but it must be remembered that healing of the changes in bone takes place slowly, several months being required for complete return to normal. Therefore, antiscorbutics should be continued for at least six months in maximum doses. Little else is to be done in the way of treatment. The mouth should be kept clean with normal saline solution or a mild antiseptic, the utmost gentleness being used in the cleansing. Styptics are rarely needed for the bleeding, which even if severe stops promptly on administration of orange juice. Separation of epiphyses should be handled by support and immobilization, and infusions into joints or elsewhere should never be evacuated. The scorbutic child should be protected from infections where possible.



## CHAPTER XIII

### NUTRITIONAL DISEASES (*Continued*)

#### RICKETS

Rickets is a chronic disturbance of metabolism; its most striking gross manifestation is bony change with consequent deformity. Known for a long time so far as the clinical picture is concerned, only within comparatively recent years have the underlying biochemical changes responsible for the pathology been understood. During the last decade the searchlight of experimental study has been focused on rickets more than any other nutritional disorder, with the result that much has been learned concerning etiology, or at least what changes take place within the tissues. To what extent these changes represent cause and effect is not entirely settled, and much experimental work yet remains to be done.

It is likely, though not certain, that the disease was known to the Greek and Roman medical writers, and medieval art shows quite clearly, during the period when realism supplanted idealism, the prevalence of this disease, since practically all the children shown in the paintings of that time are evidently afflicted. That the disease is widely distributed is well recognized, though reports from various countries differ so widely in the accuracy of the observations that it is difficult to estimate even approximately to what extent it is present in any country. It is even difficult to estimate the incidence in different parts of our own country. Much of this uncertainty arises from a lack of recognition of the disease in its early stages, before the deformity becomes noticeable. With advancing knowledge regarding the very early signs and symptoms, and the possibility of early recognition, statistical data will probably be more accurate in the future. That the disease is more common in temperate climates has been recognized for a long time, and yet observations from a number of foreign countries, particularly those in or near the Orient, show a very low incidence. In some localities there is a fluctuation in incidence, from year to year or from decade to decade. City infants are considered more prone to this disease than

are country infants, though there is no absolute proof of this statement, since records from rural localities are not available for comparison.

In large cities the disease is exceedingly common and the incidence has been variously estimated at from 50 to 90-odd per cent. These percentages are based on changes in the bones which are evident; when statistics based on recognition of the early and slightly evident signs become available it is entirely probable that the incidence will approximate the latter figure fairly closely. The infants of Italian and Negro residents in large cities are frequently affected, perhaps more so than the infants of other races. In their native countries the infants of both of these races are relatively free from the disease. It is likely therefore that the increased tendency of these races to develop rickets, when living in large centers of population, is due to the conditions under which they live, largely to insufficient sunshine. This thought is especially forceful when viewed in the light of recent knowledge. There is no doubt that rickets is far less prevalent than it was fifteen or twenty years ago. At that time almost every Negro child who attended the large clinics showed gross evidence of rickets, most of them being extremely bow-legged. To-day it is the exception to find one whose picture may be used in illustrating an article on the subject. There is a definite seasonal incidence, as is the case with several other nutritional disorders. The deformities are more pronounced in the late spring as are the more severe nervous phenomena. This is probably due to the fact that the infant has been deprived of a great deal of sunshine during the colder months. This is not a sudden development, however, at that time, but shows a gradual increase from early autumn, reaching its peak during March. Variations have also been noted during periods of prolonged lack of sunshine (rainy seasons) in the summer months. In some of the latter instances both artificially fed and nursing infants have been affected. The age incidence of rickets is usually placed between the sixth and the eighteenth months. This refers of course to the more active manifestations. Deformities disappear slowly and endure for long periods, in many cases being permanent. Recent investigations have shown that rickets may often be demonstrated during the early part of the first year by means of the x-ray. Rickets developing later than the period just mentioned has been described. For a long time such cases were denied, nor is it yet certain that they occur; some observations, however, are definite enough to cause a suspension of judgment until further investigation.

**Etiology.**—With the vast amount of experimentation during the past few years and the evidence for or against this or that theory before us, the subject of etiology is approached with much trepidation. Much has been learned, still more remains to be discovered. Theories of the etiology of rickets have been many, and attempts have been made to associate the symptomatology and pathology with conditions bearing other names. Most of the theories have been abandoned, and discussion finally limited to two theories; that relating to the diet and that relating to hygienic influences. It seems in the light of our present knowledge that both of these influences play important parts in the production of the disease.

Heredity, probably, plays no part. There is certainly no evidence to prove direct transmission; it is possible that predisposition to the disease exists which might be considered familial. It is probable also that certain prenatal influences have a part in the development of the condition. The mother's diet during the intra-uterine life of the infant may supply an insufficient amount of calcium, or predispose to its development in some other way. Premature infants are especially prone to be affected. This may be due to the fact that they are deprived of much of their normal supply of calcium since most of the calcium is supplied during the last two months of prenatal life. Under these circumstances, the infant starts its independent life with an inadequate supply of antirachitic substances stored in their tissues. Twins are also prone to the development of rickets. This may be due to a limited amount of antirachitic substances. But since both premature infants and twins (often physiologically premature) have an intense growth impulse, in many respects far beyond the normal, the development of the condition may be due to this rather than to any prenatal influence.

On account of the effect of cod-liver oil in the cure of rickets, it was quite natural that a lack of vitamin A should have been advanced as one of the theories of the etiology of this disease. Experiments on animals have proved that this particular vitamin is not involved in the etiology of rickets. It is probable that milk as an article of diet plays no part in prevention or cure. Possibly the same can be said of all other food. Clinical observation leads some investigators to the belief that infants develop rickets when fed on high carbohydrate and low fat diets.

In apposition to the food theory of the cause of rickets is the theory which relates to hygiene. Hygiene is a broad term and covers many

aspects of the manner of living. Jacobi, many years ago, stated quite positively that the main cause of rickets was faulty hygiene, or at least that it was produced by living too closely housed. He made this statement after observing the frequency of rickets in Italians and Negroes, when living in colder climates than those from which the parent stock came, necessitating living more constantly indoors. This opinion was concurred in by a number of other observers; but not until very recently has this idea been accepted as a definite if not the sole cause of this condition. Experiments have shown that infants can not only be cured by the rays of the sun and those of the quartz lamp, without any change in diet, but what is more important that the disease can be prevented by the same means. All such clinical evidence has been corroborated by animal experimentation, particularly on rats. It is quite likely that other hygienic factors play a part in the production of the disease, but in all probability the lack of sunshine is the main if not the sole cause of the development of the disease in human infants. Other theories have been advanced, such as infections, but if there is any relationship whatever, they are only a factor in lowering vital processes.

Animal experiments have shown that a diet can be devised on which rats will develop this disease. Animals fed on such a diet and kept in ordinary room light develop rickets, but when fed on the same diets and exposed to sunlight or the light from the quartz lamp, or when kept at room light and fed on cod-liver oil, the result is prevention or cure. There is a diminution of phosphorus and probably calcium. Neither cod-liver oil nor sunlight supplies these elements, but they meet the requirements of the body "indirectly, in a manner at present unknown, by so activating or altering the processes of the body as to secure a more efficient utilization of those substances which are directly or indirectly concerned with ossification and calcification."<sup>1</sup>

In treating rachitic rats with sunshine or ultraviolet ray it was found that definite evidence of healing appeared at the end of four weeks and the healing was almost complete by the end of two months. In the administration of cod-liver oil, healing was started by the end of three weeks and complete by two months.

A low phosphorus and a low calcium type of rickets have been described by certain investigators. The low phosphorus type shows a diminution of phosphorus, below 5 milligrams per 100 c.c., often

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<sup>1</sup> Powers, Park, Shipley, McCollum and Simmonds, *J. Am. M. Ass.*, Jan 21, 1922, 78: 159-165.



as low as 1 milligram per 100 c.c. This type usually shows marked bony deformity.

The low calcium type shows relatively little diminution of phosphorus, but the blood calcium is often reduced to 5 or 6 milligrams per 100 c.c. The bony deformity in this type is slight, but the tendency to tetany is marked.

Cod-liver oil has been used in the treatment of rickets for a long time. When rickets was successfully developed in rats through diet low in both phosphorus and calcium it was deduced that such deficiency was the sole cause. But rickets developed in the human infant on diet where such deficiency could not be demonstrated. In either instance the disease was cured or prevented by the administration of cod-liver oil. Such results were at first attributed to the action of vitamin A. This vitamin is especially susceptible to the action of heat and oxidation, and when cod-liver oil was subjected to oxygen at a temperature of 100° for forty-eight hours it lost its power to prevent ophthalmia. This treatment of the oil, however, did not impair its antirachitic property, which demonstrated that although the vitamin A was destroyed, the antirachitic potency was not impaired. It was then concluded that a second vitamin existed which, when fed to infants and experimental animals, enabled them to utilize calcium and phosphorus in such a manner as to protect them from the development of this disease or to cure them when the disease had developed. It is not positively determined that this substance is a vitamin, but it is so called for the present, being known as vitamin D. The substance is relatively stable and is present in abundance in cod-liver oil, but is not confined to this article. It is found also in the livers of several other fish as well as the yolk of egg and in certain plants, notably clover and alfalfa. As has been stated, similar results can be obtained by exposing the body to sunshine or the ultraviolet ray. It was shown that antirachitic properties could be transmitted to oils normally containing no vitamin D by exposing them to ultraviolet ray. It was found that this was due to the fact that through this treatment the antirachitic substance can be synthesized from cholesterol and from phytosterol. The antirachitic substance has never been secured in a pure state, but an active concentrate can be made from the nonsaponifiable fraction of oils normally containing it, or from those in which it has been synthesized through the action of ultraviolet ray. It is effective whether given by mouth, subcutaneously or intraperitoneally. The manner of its action is still unknown.

**Pathology.**—Normal bone contains approximately one-third organic and two-thirds inorganic matter. In severe rickets the proportion of these is practically reversed. All of the inorganic elements are diminished, but the greatest loss is that of calcium phosphate. In other words newly formed bone is either not calcified or is calcified to a small extent only. The changes affect all of the long bones and the flat bones, and as a result there is an unnatural softening accompanied by a varying degree of flexibility. Since the changes occur where the growth of bone is most active, the epiphyses of the long bones are noticeably affected, and as a result the bone is enlarged at this point. The lower epiphyses of the radius and tibia show marked enlargement, presenting one of the characteristic diagnostic signs.

Similar changes occur at the osteochondral junction of the ribs, causing the beading commonly called the rosary. The softening causes other deformities, such as bow-legs and bending of the shaft of a number of the long bones. These deformities are determined by a number of circumstances, such as muscular action, unnatural positions and so forth. At any prominence of a bone there may be increase in size, causing an unusual appearance, and at the attachments of ligaments or tendons nodes are apt to be formed. The flat bones, particularly those of the skull, are affected in the same manner, but the resulting deformities are frequently more striking in softening of the cranial bones from lack of phosphorus and calcium, causing the crackling feel known as *craniotabes*. Exuberant bony formation over the normal prominences of the skull bones causes bosses to form, which furnish one of the characteristic signs of this condition. All of the changes which have been described cause a variety of deformity of the whole skeleton; some extreme and grotesque examples have been recorded (see Fig. 35). In addition to unnatural curving of bones there is a compensating deposition of bony tissue, so that there is often the appearance of a markedly enlarged bone. The bones of a rachitic are generally thickened independent of the other bony changes. Fractures of long bones are common. These are usually of the greenstick variety, and when occurring synchronously with separation of any epiphysis, a relatively common accident, produce deformity which is severe and difficult to handle. On section, the bone shows an enlargement of the cartilaginous layer of the epiphysis, and the transitional zone is softer than normal and of a bluish color, rather than the usual white. There is an encroachment of the bony tissue on the red bone

marrow, this being definitely diminished. This has been said to account for some of the anemia usually seen in the severer forms of the disease. Enlargement of the heart is the rule. The bony thorax is often distorted to an extent that renders it almost unrecognizable as that of a human being. The bones of the pelvis participate in the general bony changes, producing deformities of varying degrees, and are rarely detected. Measurements of the pelvis of pregnant women disclose many such deformities; their importance in relation to infant mortality is obvious.

As has been stated, rickets is a generalized condition of disturbed mineral metabolism, presenting its most marked changes in the bones. We have observed briefly the gross appearance caused by these changes, but in order to appreciate their significance fully we must study the microscopical changes. Microscopic study reveals essentially a condition of imperfect calcification of bone, or a total absence of this process. As is well known in normal individuals, bone is formed by the cartilaginous substance between the lowest layers of cartilage cells becoming infiltrated with calcium, forming rigid columns. The vessels arising from the marrow are directed against the cartilage cells by these columns, which are in turn destroyed by erosion. These columns are partly consumed by osteoclasts, but their remains act as centers of ossification through osteoclastic activity. The new bone thus formed is known as osteoid tissue and differs from mature bone only in that it contains no calcium; when it absorbs a sufficient amount of calcium it is true bone. This process takes place so rapidly that in normal individuals very little osteoid tissue is found, and this at a very narrow zone. In rickets, on the other hand, large amounts of osteoid tissue (bone devoid of lime) are found in every part of the skeleton. This absence of calcium is particularly noticeable in the intercellular ground substance of the epiphyses. There is lacking the orderly arrangement of the vessels from the marrow, as are the bony columns from the calcifying cartilage. The cartilage does not ossify in definite order, being formed and not destroyed, as in the normal, and is therefore found in excess. In the transitional zone there is an excess of osteoid and fibrous tissue. The metaphysis is increased in diameter and in thickness, as a result of the weight it bears and the action of the muscles attached to the bone. When healing occurs, absorption of calcium by the cartilage takes place on the epiphyseal side of the metaphysis. The metaphyseal cartilage disappears and the osteoid and fibrous tissue are rapidly replaced by normal bone. Lines of calcification are found

in the rachitic zone, demonstrating attempts at healing with consequent relapses.

**Symptoms.**—In rickets as in scurvy there is a period during which the gross manifestations of the disease are developing, and during which the condition is more often unrecognized than diagnosed. Well developed cases of rickets, with marked bony deformity, are seldom observed prior to the fourth month. From this time on through early childhood, well marked cases of rickets are quite common, though as has already been stated children with marked deformity are seen with increasing rarity. The typical rachitic can hardly be overlooked, even on casual inspection. The child is usually of average or more than average weight. Many children underweight showing marked evidence of rachitic deformity are seen, but in most instances the undernutrition accompanies the rickets, and is not caused by it. Head sweating, particularly during sleep and while nursing, is an early, important and characteristic sign; restlessness during sleep is strongly suggestive. Rickets is said to cause a stationary weight in some cases, but to what extent this is true cannot be stated at this time. The head is large in proportion to the body. This is in many instances an actual relation, while in others it is merely apparent. The head has a square contour, rather than the usual rounded or ovoid contour. This is due to the formation of bosses over the prominent curves of the bones of the skull and to actual thickening of the bones. This has been explained in considering the pathology of the disease. The parietal bones and the frontals are most conspicuously affected; and when this square contour is exaggerated, there is a depression between the elevations, corresponding to the sutures, which gives the appearance of a hot cross bun. There is marked flattening at the occiput, due to pressure from the constant dorsal decubitus on the soft bones at this point. The prominence of the frontal bones often suggests hydrocephalus, from which it is not difficult to distinguish it, since the eyes do not have the downward turn, showing the whites above, so characteristic in hydrocephalus. The veins of the scalp are usually prominent even in well nourished infants and the hair is worn from the occipital region, because of the constant turning of the head. The fontanel does not close at the usual time, but remains open, often until the third year.

In younger infants, craniotabes is often found. This is, as already described, caused by the softening or failure to ossify of the cranial bones, and is elicited by alternate pressure of two fingers, when a



cause of these deformities is the lack of tone in the ligaments, and possibly also the muscles, and may result in the deformity known as genu recurvatum.

Rachitic children are often much shorter than normal for their age. Some of this may be due to deformity of the bone, but in most respects is due to the fact that the bones of the extremities fail to grow in length at a normal rate.

The effect of rickets on the eruption, general condition and tendency to decay of teeth has been much discussed. It is unlikely that enough knowledge is possessed on this phase of the disease to justify a definite statement. Some excellent observers claim that rickets delays the eruption of the primary teeth, while others claim that the disease exerts little or no influence. The condition of the teeth probably bears some relation to the presence of the disease. Deformities of the jaw, analogous to deformities of the other bony structures, have been attributed to the disease.

The muscles of rachitic infants are subject to important changes, without, however, showing a marked pathology. They are usually small and poorly developed, and on section are pale. They lack tone and power, rendering the child unable to sit alone at the normal time and to walk until quite late. The delayed power to walk is the symptom for which advice is frequently sought and brings the child for the first time under the care of a physician. For this reason rickets is not infrequently mistaken for infantile paralysis, flaccid cerebral paralysis, and congenital muscle dystrophies of various kinds. Mistakes in diagnosis are sometimes made by ex-



FIG. 65.—TYPICAL RACHITIC KNOCK-KNEES.

perienced clinicians. Constipation, the rule in marked rickets, is due to the lack of muscle power; normal peristalsis being interfered with to a serious extent. Pot belly is constant in advanced cases, and is likewise due to poor muscle power. The poor quality of the musculature of the intestines interferes with peristalsis, and as a consequence gas accumulates, causing tympany and protrusion of the abdomen. The abdominal muscles are also affected and permit the distended intestine to further balloon the abdominal cavity. Disturbance of the digestion, often with marked fermentation and gas accumulation, add to the tympany and sometimes cause an alternation of diarrhea with the habitual constipation. The digestive derangement in some instances closely resembles the clinical picture of chronic intestinal indigestion; in fact, this condition sometimes complicates the whole picture.

The picture of rickets presented is the usual classical one and is supposed to occur only after the fourth or fifth month, as already stated. The disease does not develop suddenly, but is a relatively long and insidious process. As in other diseases of disturbed metabolism there is a period during which the specific condition is developing and progressing and during which the disease is seldom recognized. With our advancing knowledge, it is possible to diagnose, or at least suspect, rickets before the classical picture presents. If we except the evidence of head sweating, beading of the ribs and that presented by the x-ray, the vast majority of cases of rickets pass unrecognized.

The x-ray findings are: "The characteristic early change occurs at the lower epiphysis of the ulna, this becomes flattened or broadened, its inferior surface is no longer sharp and straight, but concave and irregular. This surface may appear irregular and as if covered by an uneven fringe. The radius is similarly affected as the disease progresses. The shafts of the bones appear reticular and less well calcified, and the structure is evident and in too great detail. The periosteum occasionally gives the appearance of unevenness and irregularity. Development of the carpal centers may be delayed."<sup>2</sup> Beading of the ribs is demonstrable by palpation before changes at this point show in the x-ray, due to the difficulty of taking satisfactory films in this region. Sometimes changes occur also at other points, notably at the wrists, before they are noted by the ray. This is proven by subsequent examination during the course of treatment, when deposits of calcium appear as the treatment progresses. The x-ray is of especial value in demonstrating healing, and is so delicate in this regard that evidence

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<sup>2</sup> Hess, *Abt's Pediatrics*, W. B. Saunders Co.

of healing is definite on the film in many cases before there is an increase in the inorganic phosphate of the blood. "The film shows an irregular line of calcification, which soon becomes a band; later it is noted that a new tip has been added to the end of the bone. The shafts also become denser. The carpal centers ossify, or existing centers become circumscribed by a ring of inorganic salts. Finally the epiphyses become healed, the concavities become filled in, but for many months the 'scars' are visible, which inform us that rickets has been present—an abnormal contour or bowing may be noted for years."<sup>3</sup> Other bones, such as the clavicle and scapula, may also be affected and these may not be suspected until an x-ray, taken for some other purpose, discloses the altered state of calcification. The upper girdle is not, as a rule, affected with the same constancy as the lower. Fractures are frequent, all long bones being affected. In some instances the number may be so large that a suspicion of osteogenesis imperfecta may be aroused. Most cases of pronounced rickets present pronation of the feet or flat-foot, but since this is a late manifestation, and since in older children malnutrition alone may cause the condition, it is well to suspend judgment until the evidence is incontestable.

Whether we are dealing with the advanced stage of the disease, manifested by pronounced deformities, or the early stages, where bone changes are found only after the most painstaking examination, there is always present some degree of anemia, varying from the mildest form of simple secondary amenia to one so severe that one of the primary anemias is suspected.

Late rickets is a term about which much confusion has existed. Strictly speaking, the term refers to the late manifestations resulting from imperfect calcification, and are in effect more or less permanent deformities due to a previous condition. In a limited number of cases, there appears to be a recurrence of the condition after healing has seemed to be complete, or rarely in which the first evidence appears in late childhood. Cases have been reported from time to time in which the first evidence of rickets appeared in late childhood. Serious doubt existed as to the reality of such cases; since the World War, however, apparently authentic cases of late rickets have been reported from central Europe. When the bones have become calcified to an extent approximating that of the adult bone, it is difficult to understand how a process of softening can occur. Much discussion has centered on the question as to how early in life rickets begins to de-

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<sup>3</sup> *Ibid.*

velop. The x-ray has demonstrated bone changes as early as the second week. At this age, certain changes, at least suggestive of rickets, have been observed with such constancy that the additional question has been raised whether all children are affected, the condition aborting in many; or whether the changes noted in such young infants are actually rickets, or normal conditions incident to the process of bone calcification. The settlement of these points remains for the future.

It will be readily appreciated that rachitic manifestations vary in intensity and prominence from those so slight as to escape notice on physical examination, and demonstrable only by means of the x-ray, to those of so marked a nature that extreme deformity is apparent at the first casual glance. Besides the cases presenting marked bony changes, we must recognize, in the light of newer knowledge, that all children showing even slightly palpable beading must be regarded as having rickets; when to this evidence is added head sweating, the diagnosis may be considered definite. When definite alteration in the calcium and phosphorus content of the blood serum occurs, especially when accompanied by certain nervous phenomena, even where the deformity is not marked, we are taught to consider the case as one of rickets, but since there is an element of uncertainty still existing, this point will be considered separately.

**Diagnosis.**—This should present no difficulty, as a rule, even in cases not well marked. When there is an abnormal amount of sweating of the head, especially at night or while taking the bottle; great restlessness at night, especially the turning of the head from side to side often and rapidly; and a fontanel which is usually large for the age, a definite diagnosis of rickets can be made. When craniotabes and enlarged epiphyses are also present, there is no room for doubt. Muscular weakness, manifesting itself in inability to sit alone or walk at the usual age, should suggest rickets, but mental weakness must be excluded. The “curve of weakness” of the back, when the child attempts to sit alone, indicating weakness of the muscle of the back, should always suggest rickets, but we must not lose sight of the fact that a state of undernutrition without rickets presents the same sign, and that feeble-mindedness almost invariably shows the same condition. That the milder and early forms of the disease are often overlooked is quite apparent. With increase in knowledge of the subject, and an increasing tendency on the part of the physician to make more careful and detailed examination of all children coming under his care, looking especially for nutritional disturbances, the disease will be rec-



ognized more frequently in the future. A careful history and examination will make evident the diagnosis in most cases, but where doubt exists, recourse to the x-ray will be found of inestimable value. Where doubt exists as to whether a paralysis and not rickets is present, the electrical reaction will usually make the differentiation. In rickets there is hyperexcitability, while in the forms of paralysis that might cause trouble in diagnosis, there is diminution in the response.

**Course and Prognosis.**—The course of this disease is essentially chronic, or at least prolonged to an extent bordering on chronicity. Untreated cases and those in which treatment is begun late almost invariably show some permanent deformity in some part of the skeleton. The prognosis of the disease, so far as life is concerned, is good, with the exception of those manifesting marked nervous phenomena, such as tetany. The lowered state of vitality subjects the child to a number of intercurrent affections. The head sweating renders the child susceptible to frequent colds, or infections of the upper respiratory passages. Rachitic children appear to be especially subject to enlarged tonsils and adenoids, with their long train of attendant ills. The restriction of the lungs, when the thorax is much deformed, predisposes to bronchitis and pneumonia. Thus it will be recognized that, although the mortality from rickets alone is not great, the intercurrent infections are often fatal. Perhaps the most far-reaching influence of this disease is through the rachitic pelvis, causing difficult labor and consequently raising the infant death rate.

So far as the disease itself is concerned, the prognosis may be considered under two heads: cases with marked bony deformity and those with slight bony deformity. So far as we are able to judge at present, the majority of cases are of the mild type and present slight bony deformity. We may safely state that such cases probably recover without permanent bony deformity or systemic injury; though the last point cannot be determined definitely at this time. We are accustomed to consider such cases as recovering spontaneously, and yet there are a number of factors entering into the recovery which may be viewed in the light of accidental or circumstantial therapy. Cases developing in the fall or during the winter are aided to recovery by the additional amount of sunshine incident to the advent of spring, which encourages an outdoor life. The growth urge being stronger during the first year of life than subsequently is probably influential in the development of the disease; its decrease after this time also probably retards the development of the disease. To what extent the diet enters into the pro-

duction of the disease is doubtful. During the second year and last part of the first year, the diet is enlarged and extended by the addition of certain foods rich in fat, notably eggs, through which the child is subjected to certain influences which tend to retard the development of the disease, or possibly effect a cure, and which if utilized before the development of the condition would have materially aided in its prevention. Under such conditions there is effected a remarkable spontaneous cure, which includes correction of moderate deformity.

In the more severe cases manifested by extreme degrees of deformity, the return of the bones to normal contour will depend on the institution of active treatment at a relatively early period and its continuance until all evidence has disappeared. It is well known that bony deformity of rachitic origin often persists into late childhood or even into adult life. At what point actively applied treatment will prevent the permanence of these deformities is not known. To what extent this disease alone can permanently stunt growth is not certain. Those cases which appear to have been merely stunted have probably been affected with other nutritional disorders of the deficiency type.

**Treatment.**—A disease cannot be treated intelligently until the cause is known. All treatment before the cause is discovered must be viewed as empirical. It is also true that in many instances a specific has been applied empirically to the treatment of a disease; that is to say, the treatment has been discovered and used before the cause of the disease was discovered, and before it was known how the remedy acted. This has been amply exemplified in the case of rickets. There are two remedies which may be regarded as specifics for this disorder: sunshine, or the ultraviolet ray, and cod-liver oil. Both were in use before the exact nature of rickets was determined, and now all that is known is that both enable the body to assimilate calcium and phosphorus. How they act is still a mystery.

The treatment resolves itself into prophylactic and remedial. In view of the great prevalence of this disease, almost universally among artificially fed infants and to a surprisingly large extent among those who are breast fed, the question naturally arises whether all infants should not receive some form of antirachitic prophylaxis. The fact that fewer breast-fed infants develop the disease would place breast milk in the class of antirachitics; although why the breast milk acts in a manner superior to the milk of the cow is not known. The prevention of this disease, therefore, adds one more good argument for breast feeding. Hess has found that the addition of egg yolk to the

formula of a bottle-fed infant will aid in the prevention of rickets, and causes no disturbance of digestion. The two best known anti-rachitics in general use are cod-liver oil and ultraviolet ray. Each has been demonstrated as efficient when used alone, but generally both are employed. The dose of cod-liver oil advised differs considerably according to the observer and in most instances is probably too small. The demonstrations of Martha Eliot have been conducted in such a manner that it appears at this time wise to follow her suggestions. It will be noted that the dose employed by her is much larger than that generally advised. "Babies seen before the end of the first month of life are given one-half teaspoonful of pure cod liver oil twice a day. During the second month the dose is increased to one teaspoonful twice a day. If the roentgenogram shows any progress in the rachitic process, this dose is again increased, so that it amounts to one and one-half teaspoonfuls twice a day or even one dessertspoonful twice a day by the end of the third or fourth month." This work has all been done under the check of repeated roentgenographic films; and there is no reason to alter it as a routine in the general practice of the family physician. Cod-liver oil is well borne by most children. There is rarely any objection offered and in fact most infants and even children appear to like it. When there is difficulty in its administration (which I have personally never observed), it may be given in orange juice or some syrupy vehicle. Emulsions are not highly regarded by most observers and when used should be given in twice the usual dose. It is not likely that the addition of phosphorus is of benefit. Twins and premature infants should be given oil promptly after birth and in addition should be exposed to ultraviolet rays. Although much work has been done in the prevention and cure of rickets, we must not feel that the last word has been spoken, nor that cod-liver oil is quite the specific that is desired. May Wilson, in observing a series of infants born in New York, has demonstrated clinical rickets in 91 per cent of infants from one to three months of age born in the spring, and summer of 1924, who had received graduated doses of cod-liver oil from  $\frac{1}{2}$  to  $1\frac{1}{2}$  teaspoonfuls per day. In a series born in the winter of 1925 who had received doses graduated to 1, 2 and 3 teaspoonfuls daily, 68 per cent showed clinical rickets as compared with 76 per cent in control cases. A comparable per cent showed evidence of the disease when studied by means of the x-ray. The cod-liver oil used had been submitted to accurate biological tests. It would appear that hygienic measures, with the emphasis placed on sunlight, are still

the safest reliance. The other prophylactic agent is sunlight or other form of ultraviolet rays. When outdoor sun baths are given the exposure should be daily in good weather. At first the hands and feet only are exposed for ten or fifteen minutes daily for several days. For a few more days the arms are exposed for the same length of time, and the parts first exposed are now exposed for a correspondingly longer time. The exposure is increased two or three minutes each day after the effect on the skin is observed. As the weather gets warmer the whole body may be gradually exposed, and at first this should be done for an hour each day and ultimately for two hours each day. Dark-skinned infants react differently from those who are fair. The former may be exposed more rapidly than the latter. Pigmentation of the skin is a good index to the efficacy of the treatment. In the hot summer months the sun baths should be given before 10:00 A.M. and after 3:00 P.M. At this time the head should be covered. The eyes should always be protected from intense glare. Evidence is at hand that daily exposure of the arms and legs of the infant to sunshine during its daily outing is of marked benefit. In cold climates where outdoor sunlight is inadvisable or under any circumstances which make it inexpedient, artificial light may be used, and has been found to be quite effective. The mercury vapor quartz lamp is useful. Treatments are given at different distances, according to different observers. Hess recommends a distance of 3 feet, while Eliot recommends 18 inches. The exposures are made to the back and front of the trunk and to the extremities, particularly the lower extremities. The time of exposure is three minutes at first and increased two minutes at each treatment until fifteen minutes is reached if at 18 inches, and twenty minutes if at 3 feet distance. Eliot repeats the exposures two or three times a week, according to circumstances. Hess begins at weekly exposures and extends to fortnightly ones.

The carbon arc light, which gives a spectrum more closely approaching that of direct sunlight, is less irritating to the skin and may be used oftener and for longer periods at each treatment. Initial treatments are given for one-half hour at a distance of 3 feet. The arc light is especially useful in treating a number of children at the same time, as in institutions. In this case the distance may be 9 feet and the exposures longer. In all light treatments the eyes should be carefully protected.

In a consideration of the remedial treatment of rickets, the same principles obtain as in prophylaxis. When a child of six months or more is seen for the first time with the disease well developed, cod-



liver oil should be begun at once. The initial dose should be not less than  $\frac{1}{2}$  teaspoonful three times a day. This should be increased as rapidly as possible to a teaspoonful or even a dessertspoonful three times a day, according to the age and condition of the patient. In addition to this, either heliotherapy, or the light from a carbon arc or mercury quartz lamp, should be used, as in the preventive treatment. Recently experiments have shown that rickets can be treated successfully by hypodermic medication. "Cod-liver oil was saponified by means of alcoholic potash and extracted with petroleum ether. . . . The solid residue, redissolved in ethyl ether, was injected into the gluteal muscles of the patient." In this treatment the term therapeutic "unit" is employed, in which one cubic centimeter of the petroleum ether extract is equivalent to 1 c.c. of cod-liver oil. Twenty five cubic centimeters are injected in a single dose and this is repeated at weekly intervals.<sup>4</sup>

All hygienic measures should be adhered to rigidly and a well-considered formula given. In addition, other articles of food, including fresh vegetables, should be given. Infections should receive appropriate treatment and digestive disturbances corrected as soon as possible. It is generally wise to wean the infant at once if seen late in the first year. The accompanying anemia requires no specific treatment. Iron in various forms has been advised, but it is doubtful if anything is accomplished by its administration. Fresh vegetables usually furnish this element in sufficient amounts and in readily assimilable form. The deformities resulting from the disease call for orthopedic treatment, and if these are severe the child should be placed under the care of an orthopedic surgeon as early as possible. It is quite remarkable how many rachitic deformities correct themselves under routine treatment, without any special aid.

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<sup>4</sup> Wilkins and Kramer, *Johns Hopkins Hosp. Bull.*, 40: 52.

## CHAPTER XIV

### NUTRITIONAL DISEASES (*Continued*)

#### TETANY AND SPASMOPHILIA

These terms are used to designate a condition sometimes classed with the diatheses, but due, so far as is at present known, to a faulty mineral metabolism in which a deficiency of calcium plays an important part, if indeed it is not the sole cause. Many theories have been advanced for the phenomena accompanying the state; the most prominent being some derangement of the parathyroids, and a manifestation of status thymicolymphaticus. Other theories have had only passing notice. For some time this condition has been regarded as one of the manifestations of the symptom-complex known as rickets. This theory has held longer than others because of the faulty mineral metabolism found in both conditions. That spasmodic phenomena considered evidence of the spasmophilic tendency appear in the course of rickets is quite true, but the same phenomena are sometimes observed in children presenting no marked signs of that disease. The seasonal incidence of these nervous phenomena resembles so closely that of the acute manifestations of rickets that a strong suggestion is deduced that they are actually caused by rickets.

Breast-fed infants are seldom affected; on the other hand, those fed exclusively on cows' milk are most prone to develop the symptoms. The condition may be induced or caused to subside by merely changing from one diet to the other. How this change in diet so directly and promptly influences the symptoms is not understood. Whatever may be the exact metabolic changes, some interesting facts have been disclosed concerning the relation between the calcium and phosphorus content of the blood serum in rickets and in tetany. We have already observed that the normal inorganic phosphate content of the blood serum is 4 to 4.5 milligrams per 100 c.c. and that of calcium is 10 to 11 milligrams per 100 c.c. In frank rickets the inorganic phosphates in the blood serum fall to 3 milligrams per 100 c.c. or lower; while the calcium content remains normal. In cases showing either carpopedal spasm or convulsions the calcium falls to 6.0 or 7.0 milligrams per 100 c.c.

and in many cases even lower, while the inorganic phosphates remain normal. This observation inspired the thought that there were possibly two forms of rickets: the high calcium—low phosphorus rickets, which showed marked bony deformity, and the low calcium—high phosphorus rickets, which showed little or no bony deformity, but manifested a great tendency to convulsions and other nervous phenomena. Whether tetany is a manifestation of rickets or in any way associated with this disease is not at all certain at the moment. It may be that when the two are present in the same child it is merely a coincidence. Whatever may be the underlying etiological factor or factors, one fact stands out prominently, that when the calcium in the blood serum falls considerably below the normal, certain nervous phenomena occur, which we call tetany or spasmophilia, according to the clinical picture.

There are two prominent symptoms associated with the condition, carpopedal spasm, and convulsions. Carpopedal spasm is relatively rare, but presents a characteristic picture, and must be dealt with promptly. The hands are drawn to the ulnar side; the phalanges are extended and there is flexion at the metacarpophalangeal junction. The thumbs are adducted and the wrist sharply flexed. The spasm in the hand is so marked that it is only with considerable force that the joints can be moved. On the other hand, in some cases the power to voluntarily overcome the spasm is retained. The feet, while not affected as often as the hands, are in a position of equinovarus. In some instances, muscles in other parts of the body are affected by the spasms, notably the thigh. Sometimes there is evidence of pain, particularly noticed when passive attempts are made to overcome the spasm. The spasm may continue for several hours or several days, and in some cases shows a marked tendency to be intermittent. There is no less of consciousness; in fact, the patient is acutely alert. Acute attacks of tetany are sometimes observed after operation, and after digestive derangements. They are of short duration under these conditions. Tetany is frequently observed during acute infections, especially when accompanied by marked febrile reaction.

The convulsive form of this condition presents a different picture. The child so affected has a tendency to develop convulsions as a result of very slight elevations of temperature, or disturbances of digestion. These convulsions differ only slightly from ordinary epileptiform seizures, but there are several differences which call for mention. They usually begin with a tonic seizure, and pass rapidly into a clonic state. Either the eyelids or the fingers are usually convulsed

first, but in a moment the whole body participates in the seizure. There is always a varying degree of cyanosis. One point of difference between this and the epileptiform attack is that consciousness returns promptly in the spasmophilic form, whereas stupor following the epileptiform is the rule. This observation cannot be relied on absolutely, and no positive diagnosis based on it should be made.

The convulsions range in number from a single seizure to repeated attacks a few minutes apart over a period of several days. There may be only fibrillary twitchings of the fingers or toes, instead of generalized convulsions, and these may continue for several days. In tetany there appear to be two stages of calcium deficiency. I have observed on several occasions that when the blood calcium is approximately 7 milligrams per 100 c.c. that convulsions are apt to occur only as a result of some unusual stimulus, such as a moderate elevation of temperature, or a mild digestive upset. It is not at all uncommon to see convulsions occur during a mild acute otitis with moderate elevation of temperature, or where no elevation occurs. It has been observed when bicarbonate of soda has been given for a pyuria that convulsions appear to be precipitated. Such cases as these are probably in many instances cases of alkalosis. Where the calcium is reduced to a point around 4 to 5 milligrams per 100 c.c., convulsions are apt to occur without any evident precipitating cause. Other nervous phenomena are sometimes seen. Laryngospasm, spasm of the glottis or laryngismus stridulus, are terms applied to the same phenomenon. It is due to a spasm of the laryngeal muscles with consequent embarrassment of respiration. The most common form, and the mildest, is that manifested by an inspiratory crowing sound, which may continue with each inspiration or be intermittent for several hours or days. It may occur only when the child is disturbed or excited or suddenly aroused from sleep. Its occurrence is usually between the fourth and the eighteenth months. Severer attacks of spasm of the laryngeal muscles or glottis are sometimes seen, coming on suddenly and completely inhibiting respiration. There is pallor, followed by cyanosis, and sometimes by unconsciousness, but death rarely occurs. After a few moments, the spasm relaxes and the child returns to a normal state. These attacks may be the only manifestation of the disorder, or may alternate with carpopedal spasm or generalized convulsions. A condition closely allied to the preceding phenomena, if not identical in cause, is the "breath-holding spells" to which some children are prone. They may occur without any exciting cause, or only when excited by crying or



when peeved by being restrained from doing what they are intent on doing, or from fright. There is a complete arrest of any breathing effort. They sometimes endure long enough to cause pallor or even cyanosis. Convulsive movements, rigidity and sometimes generalized convulsions occur in some instances. There is usually a moment of loss of consciousness immediately following the attack. It is not clear that the cases of death reported in the course of these attacks are really due to this condition or are manifestations of some other disorder.

There are certain confirmatory signs of the presence of tetany or spasmophilia which should be sought for whenever the condition is suspected by clinical findings; these are Erb's sign, Trousseau's sign and Chvostek's sign. Erb's sign is the measure of the reaction of the nerves to the galvanic current. Normal infants and young children react to the galvanic current in a definite manner. Nerves react differently to the positive and negative poles; to the opening and closing of the current, and differently at various ages of the patient. Older children react more promptly to all electrical stimuli than younger ones. From early infancy there is a steady increase in reaction up to five years, after which the reaction is fairly constant even to adult life. In early childhood, closing contractions occur earlier than opening contractions. "In the first six months of life, any contraction with a current of less than 5 milliamperes, except that of cathodal closure, points to tetany; while an opening contraction, either cathodal or anodal, with a current weaker than 5 milliamperes, is positive evidence of tetany." This test is often difficult to elicit, and varies at different times on the same child, so that several tests should be made, before a positive diagnosis, based on this test alone, is made. Generally speaking and for practical purposes, a response to the cathodal opening current or the cathodal closing current of less than 5 milliamperes in children under five years of age is diagnostic of tetany.

Trousseau's sign is most easily elicited on the upper extremity, by pressure with the hand or a bandage around the arm above the elbow, preferably just below the deltoid sulcus. After the radial pulse is obliterated for several minutes, the characteristic carpopedal spasm appears in the hand. This sign is not constant, and its absence does not negative a diagnosis; its presence confirms the diagnosis. Occasionally a convulsion appears to be induced by eliciting this sign.

Chvostek's sign is shown by a transient and momentary contraction of the muscles of the face, notably the obicularis palpebrarum, and

the obicularis oris, or the alæ nasi, on tapping a branch of the facial nerve with the finger or the percussion hammer. Simply stroking the face over the nerve will sometimes produce the reflex. Whether these signs are pathognomonic or not is not yet clear. The younger the child the more reliance is to be placed on them. A number of other signs and symptoms occurring in the course of lowered calcium in the blood serum have been described and have been attributed to tetany. These include pyloric spasm, resulting in vomiting; spasm of the urinary sphincter and anus, causing retention or constipation; and finally spasm of the muscles of the heart, resulting in death. Proof is lacking in all of these matters and they should be regarded with skepticism until further investigation.

**Diagnosis.**—This presents little difficulty in well-marked cases where several or all of the described signs and symptoms are present. This, however, is rarely the case, hence it is exceedingly difficult to make a definite diagnosis in a large number of instances. Electrical reactions are often misleading, especially in young infants, and are frequently inconstant. The diagnosis between tetany and epilepsy is often difficult, and can rarely be made with certainty until after prolonged observation. As a rule, in young infants the convulsions are probably due to tetany; in older children the chances are in favor of epilepsy.

**Prognosis.**—This depends on a number of factors. The age of the patient and the severity of the symptoms are the two most important considerations. Young infants with severe laryngospasm, or repeated convulsions, are to be regarded with apprehension until danger is past. In older children the prognosis is fairly good. Complications, such as severe gastro-intestinal infections or derangements, and infections of all kinds, but particularly pneumonia, add materially to the chances of an unfavorable outcome.

**Treatment.**—The prophylactic treatment of the tetanic phenomena is essentially that of rickets in general. All matters pertaining to hygiene should receive most careful attention. The food should be regulated along lines which are recognized as suitable to the needs of the growing child. Children should spend as much time in the open air as possible, thereby receiving the benefit of an abundance of sunlight. The advisability of routine administration of cod-liver oil must be considered. Breast feeding should be insisted on wherever possible, since tetany rarely develops in a breast-fed infant. The discontinuance of cows' milk during the acute attacks has been advocated, but I

have seen no benefit derived from this practice. The needs of the child from a nutritional and digestive point of view should guide us in the feeding. Calcium chlorid should be administered in doses ranging from 10 to 20 grains three or four times a day, according to the age of the child and the severity of the symptoms. This should be continued so long as the indications are present. The only satisfactory manner of treating with calcium is by checking the treatment with routine examinations of the blood serum for calcium content. Simultaneous administration of cod-liver oil appears to promote the retention of calcium. All foci of infection should be removed promptly. The quartz lamp has been used with benefit. The handling of the convulsions is an important matter. All routine methods described in textbooks are of service at times, but sometimes are unavailing. The injection of a solution of magnesium sulphate deep in the gluteus muscle has proved the most reliable method of controlling the seizures. A 50 per cent solution of chemically pure magnesium sulphate is sterilized; of this, from 20 to 40 minims are injected according to the age of the child and the severity of the symptoms. The injections may be repeated as often as four to six hours, according to indications. The solution is exceedingly irritating to some children and often, in spite of scrupulous asepsis, the tissue breaks down and an abscess is formed. Even when abscesses do not form, a marked induration lasts for some time after the discontinuance of the injections.

## CHAPTER XV

### NUTRITIONAL DISEASES (*Continued*)

#### PELLAGRA

Pellagra has probably existed for a very long time; although the symptoms were observed without any idea of their significance. There is no description, however, in the literature by which the disease can be identified until that of Casal's, written in 1735 and published in 1762. It was thought at first to be a variant form of leprosy, to which the author applied the name *mal de la rosa*. Other writers of that time thought the disease had existed since much earlier times. Frapolli first applied to the symptoms already described the name pellagra, derived from the two Italian words signifying rough skin. This disease, as is obvious from the source of the earlier descriptions, was first seen and described in Italy as a relatively modern affection; but was soon described by clinicians in other parts of Europe also. The approximate date of the appearance of this disease in America is equally uncertain. There is no doubt that it was known as a symptom-complex long before any definite and accurate observations were made, and the descriptions of certain patients with unusual clinical manifestations bear out this idea. It is only since the beginning of the present century that pellagra as a definite and recognized disease has attracted much attention in this country. At present the disease is not only well known, its distribution being fairly general in this country, but the largest number of reported cases have come from the southern states, where it has existed in endemic form; but cases rather sporadic in appearance have been also reported from almost every state of the Union, and not a few have been observed in Canada. The disease makes its appearance generally during the warmer months, cool weather causing its disappearance quite rapidly. Cases do develop during the winter, however, and recurrences or exacerbations are not at all uncommon in the colder seasons. The disease is far more common in the country or semirural districts than in thickly populated areas and in cities. The poorer classes are more often affected, but cases occurring in the well-to-do classes are no rarity. It is especially common in prison camps,



asylums for the insane, and other institutions where large numbers of dependents are housed. It is in such institutions that the best and most extensive researches have been made.

Females, as a rule, are more frequently affected than males, and whites more than blacks. Insanitary surroundings appear to promote the disease, though it is doubtful if this is more than an apparent cause, since the poorer individuals live in such surroundings. There is no evidence that heredity plays any part in its incidence, and no age is exempt, though it is rare in very young infants.

**Specific Etiology.**—The history of any of the misunderstood diseases is repeated in that of another, so far as theories of etiology are concerned. Pellagra is no exception to this fact. The theories advanced have been so numerous that it would require a volume to describe them and quote the arguments offered as evidence in favor of each. Some of these theories have been ingenious and the arguments have been strong in several. Only three of these have finally been considered seriously for any length of time. The theory which for a long time was held as almost certainly proven was that the disease was due to spoiled maize or corn. It was thought that bacteria or fungi grew in this maize, and produced a toxin, the absorption of which caused the characteristic symptoms. The infectious origin of the disease was supported by an earnest group for some time. A parasite of unknown type was supposed to be transmitted by the bite of the gnat. The third prominent theory holds it to be a deficiency disease belonging in the category with scurvy and beriberi.

Each of these theories has had its ardent advocates, and there can be no doubt that there was apparently good evidence to support each of them. It is probable, however, that the theory of deficiency has more exact evidence to its credit than any other, and this theory is the accepted one at present. Excellent work has been done by a number of observers in proving the dietetic origin of the condition, but to Goldberger we owe the final proof of the specificity of a definite factor in relation to etiology, prevention and cure. The experiments conducted in an effort to unravel the tangle of theory and fact, even relating to the influence of diet alone, furnishes fascinating and instructive reading, but cannot be discussed in this brief work. The work done on rats has shown that both pellagra and beriberi can be produced or cured at will through changes in the diet. A normal rat can be made to develop pellagra; be cured of it; caused to develop beriberi and be cured of that condition within a relatively short period of time. A diet known

to produce pellagra in the experimental animal can be administered, but if to this a small amount of the pellagra-preventing factor or substance be added the rat fails to develop the disease. Exactly the same experiment may be repeated in relation to beriberi.

Human beings have also been given the same type of diets and all evidence adduced with rats is confirmed in them. The most striking piece of proof is that human beings who had had one or more recurrences of the disease on their regular diet have failed to relapse when certain articles of food or definite amounts of the pellagra-preventing factor have been added to their diet. The relation of the antineuritic factor to the development and cure of beriberi is now well known. It is the water-soluble or vitamin B. It has now been shown that diets poor in animal proteins, and even some vegetable protein, notably that contained in the legumes, almost invariably produce pellagra. It has been shown in these experiments that a definite amount of the protein of fresh lean meat (7 ounces per day) will effectually prevent the disease. It was also found that  $\frac{1}{2}$  ounce of dried yeast extract per day will as effectually prevent the disease. Also through the administration of either lean meat or the yeast extract to those who have been affected, a relapse can be effectually prevented. The efficiency of vitamin B is well known in the prevention and treatment of beriberi. The most concise statement of the facts at this moment is that of Goldberger himself: "Summing up, it may be stated that the available evidence seems to leave no reasonable doubt but that pellagra is caused by a faulty diet. The primary dietary fault appears to be of the nature of a deficiency of a factor P—P (pellagra preventing) very probably, but not certainly, identical with a dietary essential, heretofore, included with the antineuritic under the designation 'vitamin B,' which some workers have attempted to identify with bios."

In other words, the specific cause of pellagra is a deficiency of a substance or factor called factor P—P, and which is present in both lean meat and in yeast, and bears a definite relation to the growth promoting factor.

**Symptoms.**—There are three main parts of the anatomy to which the leading symptoms of this disease are referable: the skin, the gastro-intestinal tract, and the nervous system, particularly the central nervous system. Pellagra must be looked upon as essentially chronic in its course. Fulminant cases are described and are sometimes met with, but not so frequently as when the disease was first recognized. As a rule, and this is especially true in the adult, the first attack is

rather mild, and may leave the patient apparently no worse off in general health than if there had been no indisposition. There are more than apt to be repeated relapses, season after season, which leave the patient more and more debilitated after each succeeding attack. Finally, the vital forces are so debilitated that the patient is unable to regain robust health. The symptomatology varies with the individual and with each seasonal relapse. In the beginning the skin manifestations may predominate, to be followed the next season by pronounced digestive symptoms, accompanied by little or no skin lesion; while finally the mental state may be impaired and at the same time the symptoms referable to other parts of the anatomy may be insignificant or entirely absent. Nor does the predominant symptom of the first attack indicate what the symptomatic course of the disease will be nor the type which will finally prevail. The disease may be ushered in by skin manifestations, and the patient eventually die from exhaustion due to excessive diarrhea, with an entire absence of other symptoms. The frequency of one or another type and the relative incidence of symptoms have only a statistical interest and will be omitted from this discussion.

The manifestations of the disease as seen in children differ in many respects from those in the adult. It is rare that skin lesions are not the first to show, and in fact in many instances in children, remain the only indications of the disease. It is well at this point to consider the several types of symptoms somewhat in detail.

*Skin.*—The earliest symptom of pellagra referable to the skin and usually the first to make its appearance in children, is an erythema



FIG. 66.—MILD PELLAGROUS ERUPTION OF THE FEET.

This is the commonest form of the disease in children.<sup>1</sup>

<sup>1</sup>I am greatly indebted to J. Ross Snyder for all the pictures of pellagra. Fig. 69 appeared in the article by Snyder in *Abt's Pediatrics*.



FIG. 67.—DESQUAMATING ERUPTION OF FEET.

A more severe and advanced stage than the preceding.

blonde or brunette type." These changes are followed by a roughening of the skin, and finally desquamation or exfoliation ensues, commencing at the center and working outward. A brown pigmentation persists after the exfoliation, which marks for some time the area of the eruption. The skin then has a more delicate appearance over the site of the eruption than the surrounding unaffected portion, but gradually assumes the appearance of the normal skin. When the eruption has been on the extremities, a mottling of the for-

closely resembling sunburn, for which it is often mistaken. This appears suddenly, shows some degree of swelling, is sharply differentiated from the surrounding skin, and manifests no change in sensation. The eruption is of a "livid red hue," disappears on pressure, but promptly resumes its characteristics on release of pressure, and is symmetrical. Without the symmetry, no diagnosis of pellagra should be made. In from several days to several weeks, but usually in a short time, the erythema undergoes certain changes; the redness is less intense, does not disappear on pressure, and changes from the red to "brownish, bronze, chocolate, or plum shade, depending somewhat on whether the patient is of the



FIG. 68.—DESQUAMATION INVOLVING DEEP LAYERS OF THE SKIN, ULCERATIVE PROCESS.



merly affected area may persist for months after the disappearance of the eruption, when the extremities hang without support. The various stages just described require from six weeks to three months to complete the cycle. When only one attack has been experienced the skin returns to normal; after repeated attacks, however, there is a dirty, rough, and sometimes scaly appearance, which is permanent.



FIG. 69.—TYPICAL ERUPTION OF THE HANDS OF MODERATE SEVERITY.

The description of the eruption just furnished is that of what is usually termed the dry form. This is by no means the only form, though it is most frequently observed in children. There may be vesicles or bullæ which contain serum, or sometimes blood, causing oozing of fluid which varies according to the contents of the vesicles. When these lesions become infected ulceration may occur, and this complication renders the prognosis extremely grave.

As a rule, the exposed skin surfaces are affected: the extensor surfaces of the hands and feet, sometimes extending up the arm or leg. The face is often affected, particularly under the eyes (butterfly type), while there is sometimes seen a collar of eruption around the neck. In many children, particularly those who go barefoot, the eruption may not be noticed, and in mild attacks the whole condition escapes



FIG. 70.—MODERATELY SEVERE ERUPTION OF HANDS. ULCERATING AND BLEEDING BULLOUS ERUPTION OF FEET.

attention. Occasionally the eruption becomes general in young children, but rarely goes beyond the erythematous stage.

*Digestive.*—These differ to a considerable extent from those observed in adults. The sensations referable to the mouth, esophagus and stomach are common to both. The mucous membrane of the mouth is fairly constantly affected with an inflammation. In some children this is so slight that it is not complained of at all. There is, however, usually a sense of dryness or burning, and this often extends down the esophagus and into the stomach. The inflammation of the mouth varies from slight congestion with roughness to marked stomatitis with ulceration. The tongue may be furred, with red edges or it may be uniformly inflamed and even swollen to a marked extent. There may be excessive ptyalism, and the gums often resemble a scorbutic process, swollen, and tending to bleed. The extreme anorexia of the adult type is rare in children, so that those with severe stomatitis eat well, and thereby hasten recovery. Diarrhea is not the constant manifestation in children that it is in the adult, constipation being quite common. Dysentery is not rarely observed, but whether this is a manifestation of the disease or an intercurrent affection, induced perhaps by a lowered states of nutrition and general health, is not certain.

*Nervous System.*—The marked pain and tenderness often complained of by adults is rarely observed in children. Exaggerated reflexes are sometimes seen, and in those showing this phenomenon, pain may be manifest. The headache and other sensory symptoms of the adults are not present as a rule in children. Delirium is said never to occur in children. Some drowsiness is present during the acute stage, and changes in disposition are sometimes noted.



FIG. 71.—A WRETCHED PELLAGRIN, WITH BULLOUS ERUPTION OF HANDS AND SCATTERED ERUPTION OF FACE.



FIG. 72.—BLEEDING ERUPTION OF FEET.  
CASAL'S COLLAR PLAINLY SHOWN.

The fulminant type of the disease is not seen in young life. Death rarely occurs, and in general the disease runs a mild course and is easily controlled.

**Treatment.**—The treatment of pellagra is largely dietary and hygienic. Drugs play no part in the treatment of this disease. Many of these cases do much better if removed from their homes and treated in institutions, in order that the diet may be properly controlled and the child be put in the best possible hygienic surroundings. If such a course cannot be carried out, hygienic conditions in the home should be improved as far as possible. Parents should be instructed as to the necessity of abundant fresh air, cleanliness, rest and exercise. Oral hygiene should be especially stressed.

**Diet.**—A *well-balanced* diet, such as many children receive, will successfully protect against pellagra. Milk, meat and eggs in sufficient amounts will prevent and cure this disease. Goldberger would include legumes, such as peas and beans. Yeast or yeast extract has been found by Goldberger<sup>2</sup> to be very effective in the treatment of severe cases, but unnecessary in the

milder types. The well-known yeast cake may be stirred up in any convenient medium, such as water, milk, orange juice or tomato juice.

<sup>2</sup> Personal communication from Goldberger.



Dried baker's yeast or dried brewer's yeast may be used in the same way. There is also a dried yeast extract on the market which is a concentrate of the pellagra-preventive factor, and which is more potent than dried yeast. One-half to one ounce of dried yeast has been found sufficient. If moist yeast is used, the dose should be five or six times that of the dried yeast. If the yeast tends to produce flatulence, this may be avoided by bringing the yeast suspension quickly to a boil. In every case concomitant disease must be properly treated if we are to make much progress. The author agrees with Snyder that a pellagrous mother be not allowed to nurse her infant. The character of her milk

is apt to be impaired and the strain of nursing an infant will prove a drawback to the mother herself, materially interfering with the treatment.



FIG. 73.—BUTTERFLY MASK TYPE OF ERUPTION AS SEEN IN A MULATTO.

## CHAPTER XVI

### NUTRITIONAL DISEASES (*Continued*)

#### BERIBERI

(*Endemic Multiple Neuritis*)

Beriberi is classed among the deficiency diseases and it has been definitely proven that in the Orient at least this disease is caused by a deficiency in the antineuritic or so-called water-soluble vitamin B. The diet largely responsible for this disease in the Philippines and other eastern countries where beriberi is endemic is a diet of polished rice. Fraser, Stanton, Strong, Crowell and others have demonstrated that the disease can be produced in humans fed on such a diet. Eijkman, in a striking series of experiments on fowls, found that a polyneuritis similar to the disease as it occurs in man could be produced by feeding a diet consisting exclusively of polished rice. He proved further that the disease could be cured by feeding affected animals the polishings of rice.

Extensive studies followed this notable work of Eijkman and it was found possible to extract this protective substance from rice polishings. Such an extract will not protect against beriberi, but it will cure after the disease has developed. As the result of such investigations, it has been possible to prevent the occurrence of the disease in many institutions and communities where beriberi was once prevalent. So simple a thing as changing from a dietary consisting largely of polished rice to one of undermilled rice has made this possible in countries where rice constitutes the main article of diet. Among the other foods deficient in water-soluble vitamin B are highly-milled wheat flour and the various sugars. Whole wheat, whole rice, barley and beans contain the vitamin in amount sufficient to protect against beriberi.

Not alone is the adult susceptible to beriberi, but also are infants and children.

The work of McLaughlin, Andrews, Chamberlain, Vedder, Williams and others has proven very definitely that there is an infantile type of this disease and that it is a very frequent cause of death among infants in Oriental countries. Native physicians of the Philippines

recognized this disease, but did not associate it with beriberi of adults. It was left for Andrews, Chamberlain, Vedder, Williams and others to advance and prove the theory that infants nursing a mother suffering from beriberi might in turn develop the disease. Andrews fed puppies on milk from mothers whose infants had died from infantile beriberi, and on autopsy the puppies showed similar lesions to those of the infantile type of the disease. Vedder and Williams showed that infantile beriberi could be cured by feeding an extract of rice polishings. Cures resulting from this method of therapy were as spectacular as those seen in scurvy treated with orange juice. From the above works, it seemed clear that milk from mothers suffering from beriberi was itself deficient in the antineuritic vitamin.

"Infantile beriberi is an acute or subacute disease affecting infants who are nursing mothers suffering from beriberi. The disease is characterized by cardiac hypertrophy, generalized edema, chronic passive congestion of the liver, kidneys and spleen, and punctate subpleural hemorrhages, together with a certain degree of peripheral neuritis. The clinical picture is that of cardiac disturbance, with dyspnea, gastro-intestinal derangements, edema, oliguria and aphonia. It is important to remember that there is no essential difference between beriberi in adults and beriberi in infants, except for the fact that, as the infant neither walks nor talks, it is impossible to elicit the various motor, sensory, and subjective symptoms that are obtained from the adult."

**Pathology.**—Right side enlargement of the heart is the most constant finding at autopsy. There is a tendency towards generalized edema and considerable amount of fluid may be found in the pericardial sac, peritoneal and pleural cavities. There is a generalized congestion of abdominal viscera, and a degenerative inflammation of peripheral nerves.

**Symptoms.**—Vedder describes two types of infantile beriberi: (1) the acute pernicious or cardialgic type; (2) the chronic type. In the acute type the onset is sudden, the infant apparently being in considerable pain and constantly crying. Examination of the chest shows the heart enlarged toward the right. One attack follows another, death being a matter of a few minutes or hours and is cardiac in origin. In the chronic type, the first symptoms to attract attention are a rather obstinate constipation and vomiting. There is usually morbid fretfulness. Edema of the extremities associated with a diminution in the amount of urine passed soon develops. Aphonia is not an uncommon

symptom and is thought to be due to changes in the pneumogastric nerve. As in the acute type, there is enlargement of the heart to the right. If allowed to go unheeded, death is the usual result.

Vedder stresses the importance of a careful history in old cases, calling attention to the fact that the condition occurs in nursing babies under four months of age. The symptoms of the disease in the mother may be slight or very apparent, but are always present.

**Treatment.**—Drugs play no part in the treatment of this disease. Extract of rice in suitable doses, introduced in the Philippines by Vedder and Chamberlain, has proven almost specific in the hands of these and other workers. This treatment must be kept up for several weeks and has the distinct advantage that it does not necessitate taking the infant from the breast.

**Prophylaxis.**—Feeding of articles of food rich in the antineuritic vitamin, such as peas, beans, carrots, eggs, milk and meat, will prevent the occurrence of the disease in mothers, thus protecting the infant. Extract of rice polishings, while more or less of a specific, has not been used to a great extent, due to ignorance of the natives of the countries in which the disease is present and the failure to have proper legislation passed.

*Method of Preparing Extract of Rice Polishings (Vedder).*—The rice polishings consist of all the materials removed from the rice. "The polishings are sifted, weighed and extracted with 90 per cent alcohol. During extraction the alcohol becomes deeply tinged with green by the fat which is dissolved. The extraction may be repeated with several portions of alcohol. The alcohol so obtained is filtered and evaporated on a water-bath at not higher than 80° C. An electric fan facilitates the evaporation. When the alcohol is entirely evaporated, the residue is poured into a separating funnel and allowed to stand for about an hour, at the end of which the liquid will separate into two layers. The upper and larger portion, which is deep green, is the fat; the lower layer is brown in color, of syrupy consistency, and contains the vitamins that have been extracted, together with sugar, resins and other substances. As the vitamins were found to be soluble in water, this lower layer is carefully drawn off and diluted with water about twice its bulk, whereupon a heavy precipitate of resins and other similar substances soluble in alcohol, but not in water, is formed. This precipitate is filtered off, and the clear brownish fluid is the extract of rice polishings, ready for use."

In Reprint No. 1062 from the Public Health Reports, February



19, 1926, pages 12, 13 and 14, the following statements are made: When young rats are fed a diet complete for growth except as to the "water-soluble B," but containing as the sole source of this vitamin as much as 40 per cent of a preparation of an alcoholic extract<sup>1</sup> of corn meal that can alleviate or cure polyneuritis in the rat, the weight of such animals, after slight initial growth, is arrested and then declines. If, however, when signs of polyneuritis begin to appear, there be included in "certain diets" as little as 5 per cent of our alcoholic corn extract (40 per cent of which as the sole source of water-soluble vitamin in a diet does not enable the rat to grow), the animals, if not too far gone, recover from polyneuritis and resume growth.

"With respect to the specific question . . . relative to the preparation of the alcoholic extract of corn meal, . . . I desire to state that this preparation was devised for purely experimental research purposes. We have not used it in human beings, but it doubtless would serve in the cure and prevention of human beriberi. However, as it is a somewhat tedious and laborious job to prepare this extract, it does not seem to me that it should be considered seriously for use in the human disease—more particularly as a good dried yeast is more readily available, cheaper and, for the particular purpose, as efficient, or even more efficient, in the same dose."<sup>2</sup>

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<sup>1</sup> This extract is prepared by intermittent percolation of whole white corn meal at room temperature with alcohol of 85 per cent by volume strength, until about 6.5 liters are obtained from 5 kilograms of corn meal. The percolate is put into a distilling flask and concentrated to about one-fifth to one-fourth its volume. This is then poured into a pan on a water-bath and cornstarch stirred into it at the rate of 125 grams of starch to 5 kilograms of corn meal used. The remaining alcoholic liquid is driven off by fanning. The damp residue is then transferred to glass dishes and further dried in a current of warm air, after which it is ground into a powder. For each 18 to 18.5 grams of corn meal 1 gram of this product is thus obtained.

<sup>2</sup> Personal communication from Goldberger.

## CHAPTER XVII

### ACIDOSIS, ALKALOSIS

#### ACIDOSIS

There are few more emergent situations than the disturbance of nutrition known as acidosis, an abnormal process which depletes the fixed alkalies of the body. During normal metabolism various kinds of acids are produced, some of them in large amounts. The acids produced are carbonic acid, phosphoric acid, and sulphuric acid, and besides these various organic acids. Normally the body eliminates these acids or neutralizes them with ammonia, so that a balance is maintained and no depletion of the fixed alkali reserve in the blood and tissues occurs. The balance is disturbed in several different ways: through the excessive production of acids; through the excessive intake of inorganic acids; when the intake of bases is insufficient to meet the requirements; when there is a disturbance of elimination; and when there is a failure of the mechanism of neutralization. When any of these factors are deranged, a process known as acidosis is inaugurated, causing a depletion of the fixed alkalies of the body. This depletion produces a definite train of symptoms, to which the name acidosis is likewise applied.

The blood plasma and the tissue juices are so closely analogous in composition that, for all practical purposes, they may be considered as one and the same. The plasma is an alkaline fluid depending for its alkalinity on the presence of protein, sodium bicarbonate and inorganic phosphates; the most important of these being sodium bicarbonate. In our consideration of plasma, it is regarded as a 0.3 per cent solution of sodium bicarbonate. As the blood circulates through the tissues, it absorbs a considerable amount of carbon dioxide, a product of metabolism. A large amount of this substance is absorbed as solution, before it affects the even slight alkalinity of the plasma. Gradually and finally as more carbon dioxide is dissolved in the plasma, the alkalinity becomes less and less, until the plasma is distinctly acid. The changed reaction, however slight, is sufficient to stimulate the respiratory center, with greater activity on the part of the muscles of respira-

tion, thus eliminating the excess of carbon dioxide through the lungs. Since metabolism is increased, the excessive supply of carbon dioxide is continued, and the unusual stimulation of respiration becomes sustained.

As a result, the respirations become quicker and deeper, and successfully eliminate the excess of carbon dioxide, and no depletion of the alkali reserve takes place. This is familiar to all as a result of extraordinary exercise.

A similar condition may be produced in another manner through abnormal metabolism. This latter is a more serious condition, since the processes are more involved. When certain abnormal processes of metabolism take place, a number of nonvolatile acids are formed in quantities in excess of the normal production, and are taken up by the plasma, during the circulation of the blood through the tissues. The acids liberated under these conditions are  $\beta$ -oxybutyric, aceto-acetic and lactic acids; and when they reach the plasma and are absorbed they act on the sodium bicarbonate and liberate carbon dioxide. The plasma reaction is then, of course, changed from alkaline to acid, with the result, as in the other process, that the respiratory center is stimulated, and an effort is made through excessive respiratory activity to get rid of the excess carbon dioxide. In the present instance, however, a large proportion of the bicarbonate has been used, by depriving it of the amount of carbon which was required to generate the carbon dioxide, when the acids acted on the bicarbonate. This partially depleted plasma bicarbonate then absorbs more carbon dioxide from the tissues through which it circulates and the acidity is increased; the plasma being already impaired as to its alkalinity. Thus the respiration is further stimulated, but the vital capacity of the lung is unable to exhale the excessive carbon dioxide. The production of carbon dioxide is not equal to the increased work put upon the lungs and thus the carbon dioxide "tension" in the lungs is diminished, usually one-half. These facts indicate that the bicarbonate reserve in the blood is diminished one-half and the pulmonary ventilation has been doubled.

The lungs have done their part in an attempt at stabilizing the alkali balance of the body; they have not been equal to the task alone. The body now further comes to their aid and generates an increased amount of ammonia, which combines with the acids and forms bases, which serve to neutralize the acids in the plasma, and some of these combined bases are excreted in the urine, largely as acid phosphate.

The amount of ammonia salts in the urine (excessive) is one of the laboratory evidences of the presence of acidosis.

All of the usual efforts of the body functions may be called into play in order to overcome the state of acidosis, and yet they may not be equal to the task; and so artificial aid must be evoked. The treatment of acidosis will be outlined later in this discussion.

**Symptoms.**—The first symptom to be noticed is a change in the respirations. These are deep and heaving and made with decided effort. The rate is unaffected. The breathing differs from that of any other condition; it has not the rapid and shallow character of pneumonia, nor the labored and noisy breathing of any form of obstruction. It is deep, continuous and silent. There is no cyanosis, except in those rare cases of acidosis accompanying cardiac disease. Cherry red lips and cheeks have been emphasized as of diagnostic value. This leads to error, since such signs are not constant, and have little or no significance either way. An acetone odor to the breath has likewise been emphasized as of importance; such an odor is present only in acidosis, due to the acetone bodies. Drowsiness or even stupor are sometimes, but by no means constantly, present.

There are a number of clinical types of acidosis which correspond to the pathological change, or to the acid or other bodies causing the symptoms. The type which represents the largest number of cases is that due to acetone bodies. Whenever an insufficient amount of carbohydrate is metabolized simultaneously with fat, incomplete metabolism of the fat occurs, so that its catabolism stops at the stage of the acetone bodies, that is, acetone, oxybutyric acid and aceto-acetic acid. The two acids mentioned produce acidosis, in the manner already described. During starvation, when stored fat is drawn upon, acetone bodies are excreted in the urine in large amounts. This rarely results seriously. In febrile conditions, there is usually an increase in basal metabolism and an increase of acetone bodies results. In all infectious processes, a similar condition is produced, due probably to the lowered power of the digestion and metabolism. The increase of acetone bodies seen in diabetes mellitus is probably due to a failure of the body to utilize carbohydrates. Postoperative acidosis is well recognized, and is difficult to explain. It may be due in part to excessive vomiting, though this cannot account for all the cases, since it occurs after some operations, where vomiting does not occur. The ether itself may cause the condition, but this is not as yet proven.

A condition still imperfectly understood is cyclic vomiting. This



condition is usually accompanied by the presence of acetone bodies in the urine and also by an acetone breath. In some cases, these bodies are found in the urine within a very short time after the beginning of the vomiting, and are sometimes present in large amounts. In several cases observed by the author, the acetone bodies appeared in the urine from one to two days prior to the onset of the vomiting. Whether the acidosis is due to the formation of these acetone bodies, or is merely an accompanying manifestation, is still a debatable point.

Lactic acid is produced in excess in a number of conditions, such as excessive exercise, convulsions, surgical shock, phosphorus poisoning, and acute yellow atrophy of the liver, and may lead to or be accompanied by acidosis. An acidosis, supposed to be due to an unknown organic acid, is often observed in lobar pneumonia. The acidosis occurring in the course of cardio-respiratory disease is supposed to be due to an excess of carbon dioxid, and also possibly the excess production of lactic acid.

We have seen that the kidney has the power of excreting certain acids, notably phosphoric acid in the form of acid phosphate. In the course of renal disease, there may be a failure on the part of the kidney to excrete this substance and, as a result of its accumulation, an acidosis may occur.

Diarrhea is well known as a condition in the course of which a severe acidosis may develop. This is probably due to an excessive elimination by the bowel of sodium salts, though this theory is disputed, because there is also an excretion of bases. It may be due to failure of the kidneys to excrete the acids, or to a retention of certain products of metabolism resulting from incomplete oxidation. The exact cause remains unknown.

Acidosis has been noted as a result of excessive ingestion of certain acids; it also occurs in cases of poisoning by a number of substances.

An acidosis is frequently observed in cases of burns and also in surgical shock, and occasionally in severe hemorrhage. To what this acidosis is due is not known. It is more likely that it is an accompaniment, rather than either a cause or an effect. The diminished flow of the blood which results from all of these conditions is the most likely explanation.

**Laboratory Tests.**—The clinical diagnosis of acidosis offers no particular difficulty to the trained observer; regardless of this, the diagnosis should be checked by laboratory tests, of which there are sev-

eral; none very difficult. The first is the test for carbon-dioxid tension of alveolar air. This test is most conveniently made by using the Marriott apparatus.<sup>1</sup>

The carbon-dioxid alveolar tension varies in normal children at rest from 40 to 45 millimeters. A tension from 30 to 35 millimeters indicates mild acidosis. When the tension reaches as low as 20 millimeters, the condition is critical. Lower readings are found during coma. In infants, the tension is uniformly from 3 to 5 millimeters lower than the above figures.

A number of conditions render observations on carbon-dioxid alveolar tension unreliable. Among these are disorders of the respiratory center; the previous administration of certain drugs, notably caffeine and its class; intracranial lesions; and in diabetes, after treatment with alkalis. Disease of the mucous membrane of the bronchi, which interferes with a free exchange of air with the blood, will also often vitiate the readings.

A second test for the determination of the presence of acidosis is the Van Slyke method, which indicates acidosis when the alkali of the plasma is lower than 40 volumes per cent.

A third and quite convenient test is one by which the alkali tolerance is measured. This test depends for its value on the fact that when the bicarbonate content of the plasma is within normal limits a very small amount of sodium bicarbonate by mouth changes the reaction of the urine. The excess of bicarbonate is excreted and the normally acid urine becomes slightly alkaline or amphoteric. During acidosis, the bicarbonate content of the plasma being low, a larger amount of bicarbonate by mouth is required to effect a change in the urinary reaction. In normal infants, 1 to 2 grams of soda are sufficient to produce the change; in acidosis an increase up to ten times this amount is required. Litmus paper is unreliable for the test, the most delicate being cresol purple;<sup>2</sup> a drop of the indicator is added to a specimen of freshly-passed urine; if the color changes to violet or purple, acidosis is not present; if it remains green, acidosis may or may not be present. Alkali should then be administered by mouth, until the urine just causes a purple coloration of the indicator.

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<sup>1</sup>Apparatus made by Hynson, Westcott and Dunning, Baltimore. Instructions for its use accompany the outfit.

<sup>2</sup>Solution for this purpose, prepared by Hynson, Westcott and Dunning, Baltimore

**Treatment.**—"The treatment of acidosis is to be based on three principles: the prevention of further acid formation; replenishing the alkali reserve; and the elimination of acids and other salts." To accomplish these, there are two main methods of treatment.<sup>3</sup>

*The Glucose Method.*—Glucose may be administered by mouth, either as a simple solution or flavored with orange juice. For this purpose a 5 to 10 per cent solution of glucose is administered, depending on the age and condition of the patient. This may be administered in varying quantities, at intervals of two hours or more. In mild cases, it is often quite satisfactory and it is unlikely that an excess of glucose is administered in this way. The rectal administration is by means of the drip method, using a 5 per cent solution. In this way at times a large amount may be used, but the mucous membrane of the rectum is easily irritated by the glucose and after a while expels the solution as rapidly as it is administered. For rapid administration, the intravenous route may be employed. In this way 5-10 per cent solution, depending on the degree of acidosis and age of the child, may be used to the extent of 20 c.c. per kilogram of body weight. This is to be administered very slowly. Since it is often difficult to enter the veins of children, especially the very young, the intraperitoneal route will be found eminently satisfactory and harmless. The same solution may be used as by the venous route, though larger quantities may be given, since the absorption is relatively slow, and it is not necessary to give it slowly.

*Alkali Therapy.*—Where depletion of the alkali reserve has occurred to a marked extent, alkalies are indicated. Sodium bicarbonate is used for this purpose. Where intravenous administration is to be employed, it is wise to calculate the exact amount needed. This is a fairly simple procedure. "The blood and tissue fluids contain approximately 3 grams of sodium bicarbonate per kilogram. As approximately 70 per cent of the body weight is made up of such fluids, the total amount of bicarbonate in the body is approximately 2 grams per kilogram of body weight. If the blood bicarbonate or the alveolar carbon-dioxid tension is diminished one-half, then it is necessary to administer 1 gram of bicarbonate per kilogram of body weight to restore normal conditions." Either of the following formulæ may be employed, depending on whether the carbon-dioxid alveolar tension has been used or the bicarbonate reserve of the blood plasma.

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<sup>3</sup> Adapted from the work of Howland and Marriott.

$$\frac{40-T}{40} \times 2W = \text{grams of sodium bicarbonate needed}$$

$$\frac{60-V}{60} \times 2W = \text{grams of sodium bicarbonate needed}$$

In each formula

T = alveolar carbon-dioxid tension in millimeters.

V = volume per cent carbon dioxid in plasma as determined by Van Slyke method.

W = body weight in kilograms.

It will be observed that this calls for a rather large amount of bicarbonate of soda to restore the balance of alkali reserve. Where the sodium solution is given intravenously, a 4 per cent solution is used in amounts varying, according to indications, from 15 to 20 c.c. per kilogram of body weight. This is to be administered slowly several times within the twenty-four hours. To prepare the solution, the soda is added to freshly distilled and sterile water. No further sterilization is needed. By mouth 1 to 4 grams may be administered every two to four hours, well diluted. When bicarbonate of soda is used for this purpose, it should be obtained from a fresh, sealed package of the C.P. product. When it is not feasible to make laboratory tests for carbon-dioxid tension of blood plasma content of bicarbonate, the following simple test may be used. "Drop of cresol purple is added to a fresh specimen of urine. If the color remains green more alkali is needed. If it changes to any shade of red or purple, a sufficient amount of alkali has been given."

*Water Administration.*—Regardless of whether we use glucose or soda, the importance of the administration of water must not be lost sight of. In fact, it is more important than either and many cases, if not too severe, can be controlled by water administration alone. Where no vomiting exists, water may be administered by mouth, prescribing definitely the amount to be used in twenty-four hours. This varies from 30 ounces in infants to 125 in older children. Where it is difficult or impossible to induce the child to drink water (an experienced nurse can usually accomplish this), we may resort to the stomach tube, absorption of water by this route being rapid and satisfactory. In many cases, however, it is impossible to accomplish much in this way. Hence intraperitoneal administration is exceedingly satisfactory. In infants during the last half of the first year of life,



from 150 to 250 c.c. of normal saline solution may be administered by the intraperitoneal route as often as every six or eight hours. In older children from 250 to even as much as 400 c.c. may be administered.

## ALKALOSIS

The condition known as alkalosis has occupied a rather prominent place in the medical literature of the past few years. The means by which the acid-base relation is kept constant in the blood stream has been discussed in the previous section on acidosis. This is brought about by so-called "buffer" materials, chief of which are the salts, hemoglobin and proteins of the blood stream. In the condition known as alkalosis, the reaction of the blood stream becomes more alkaline than is normally the case. This change toward the alkaline side is due largely to soda bicarbonate, the phosphates and proteins.

The most frequent cause of alkalosis is the administration of soda bicarbonate or other alkalis for therapeutic purposes. This is quite a popular form of therapy with many practitioners and is not without danger. Convulsions and other symptoms of spasmophilia may result from such therapy. MacCallum and McCann have observed alkalosis in dogs in which artificial pyloric obstruction has been brought about. It would seem that alkalosis in such cases was due to loss of hydrochloric acid as the result of vomiting. Might not the same condition result in infants suffering from congenital stenosis of the pylorus or persistent vomiting from any cause, particularly if solutions of bicarbonate of soda were given for therapeutic purposes. Cases of alkalosis have also been reported following the use of the Roentgen ray for deep therapy and in high fever from various causes. In the high fever cases, overventilation of the lung would seem to be an important factor, both rapid and deep respiration.

Symptoms of this condition are variable, and alone do not warrant a diagnosis of alkalosis. The diagnosis must be made in the laboratory, blood bicarbonate and PH determinations furnishing the necessary proof. Some of the symptoms are headache, nausea and vomiting, fever, tremors, convulsions and other evidence of spasmophilia.

This condition is difficult to treat when once established. All alkalis should, of course, be withheld and dilute hydrochloric acid or ammonium chlorid administered by mouth.

## CHAPTER XVIII

### SWIFT'S DISEASE (ACRODYNIA; ERYTHREDEMA)

Under the name of acrodynia,<sup>1</sup> erythredema and a number of others, has been described a condition with symptoms referable to the nervous system, the skin, and nutrition, whose etiology and classification still remain in doubt. Theories of etiology are numerous, each report of cases having been accompanied by some discussion of etiological factors, with emphasis on one particular theory which, according to the particular author, would appear to throw light on the syndrome. Reference to the literature as far back as a hundred or more years discloses accounts of a condition occurring both sporadically and in apparently epidemic form coinciding with our present knowledge of the disease sufficiently to justify the assumption that it was the disease under discussion. Most of the early reports were made on the continent, chiefly in France. When Swift, in 1914, described a condition before the Australasian Medical Congress similar, if not identical with, the previously reported cases, and designated the condition clinically as erythredema, the attention of medical men throughout the world was attracted, and since then a large number of cases have been reported. From various parts of the United States and Canada have come reported cases with interesting and concise descriptions of the clinical findings. The cases reported have varied from isolated instances to a considerable number occurring within a limited area, and within a short period of time.

The term acrodynia literally means pain in the extremities, and originated from one of the symptoms. Erythredema was applied because of the redness of the hands and feet, though this was not accompanied by a pitting edema. "Polyneuritic syndrome" has been employed because of certain nervous phenomena, and it is interesting to note that a number of writers have used the expression "a pellagrous-like affection"; and too there has been a constantly recurring expression of opinion that the condition was a variant form of pellagra. The term

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<sup>1</sup>For our knowledge of Swift's disease as it occurs in this country we are indebted to the observations of J. B. Bilderback, Albert H. Byfield, Frank H. Lamb and William Weston.

"Swift's disease" is used in this discussion in response to the suggestion of Warthin in a recent article (*Arch. Path. & Lab. Med.*, Vol. 1, No. 1), in which he reports autopsies on two cases.

**Symptoms.**—The onset varies greatly in different individuals and in different outbreaks. It may be either sudden or gradual. Change in disposition is first noted; fretfulness and peevishness being marked. Restlessness during sleep, which is much disturbed, is prominent, and the appetite varies from slight reluctance to the taking of food to complete anorexia. Regardless of the character of the onset, the skin manifestations are not long in appearing. "The feet and hands become red and swollen, accompanied by intense itching or burning of the palms or soles. A rash may also appear on the tip of the nose, the ears and cheeks, or it may cover the entire body except the back of the neck. Soon after the rash appears desquamation begins. The desquamation sometimes involves all the layers of the skin and deep necrotic ulcers form. Usually, however, it only involves the superficial layers, leaving a bluish red glistening surface, giving a cold and clammy sensation to the touch. These areas may be either hyperesthetic or anesthetic." Throughout the period of skin changes there is profuse sweating, which appears to be irritating. Pallor and emaciation are prominent; photophobia is marked. Muscle power is much reduced. The child refuses to walk, but whether this is attributable to weakness of the muscles themselves or is due to involvement of the peripheral nerves, or to both, cannot be stated definitely. The feet are rubbed against each other, or against the bed clothes or other objects, until they become raw. The child presents a pitiful picture of abject misery.

The skin manifestations on the extremities are usually uniform, but



FIG. 74.—ERUPTION OF NOSE AND CHEEKS IN SWIFT'S DISEASE.<sup>2</sup>

<sup>2</sup> Photograph of colored illustration of the very excellent article on this disease by Albert H. Byfield, *Am. J. Dis. Child.*, 20: 347.

not constantly so on the trunk. The fingers and, to a less extent, the hands, the toes and also the feet, are practically always involved. These manifestations may remain constant throughout the course of



FIG. 75.—ERUPTION ON HAND IN SWIFT'S DISEASE.

the disease, or may be remittent. Sometimes the skin in these localities is cold and cyanotic. The skin involvement is not sharply defined, but the affected area gradually fades into the normal skin.

When the skin of the trunk is involved there is more apt to be a mottling rather than a uniform redness.

Paresthesias of fingers and toes are constant. There is an almost constant desire to pull the hair out, which the patient does frequently, leaving a number of bare spots. The hair itself, which is unusually dry, drops out over well-defined areas, resulting in the same appearance.

Photophobia is common, and certain changes in the cornea suggest ulceration, or a process suggestive of food deficiency. There may be some glandular enlargement. The teeth often drop out without

local manifestation. Diarrhea is sometimes present. The reflexes are not constant; being normal, diminished or exaggerated in individual cases.

A number of complicating infections have been described, to each of which has been ascribed etiological significance. There is nothing definite on this point as yet.

**Etiology and Pathology.**—As has been hinted already, this condition has been attributed to a variety of etiological possibilities. It is quite natural, from the various clinical manifestations, that infec-



tions and food deficiencies have been the two most constantly considered. Focal infections, especially those of the nasopharynx and the tonsils, have been studied in relation to the etiology, but so far nothing definite has resulted from this line of thought. In one series of cases, there was a nasopharyngeal infection present or at least preceding the onset of the disease. In another series, improvement was noted after the removal of the tonsils. The suggested analogy to certain stages and symptoms of pellagra has caused many to regard the condition in the class of deficiency diseases, if not pellagra itself. The fact that in most cases observed no defects in the diet have been discovered has discredited this theory to some extent. That it is not always easy to discover deficiency in the diet is well known to students of other deficiency diseases, and so this theory cannot be dismissed at this time, any more than any other one can be.

The report by Warthin (already alluded to) of autopsies on individuals dying because of or in the course of an attack of this ailment sheds much light on the pathology, and strongly suggests a rational theory of etiology. Warthin's report will be quoted in its entirety:

"Acute infection of the respiratory tract was present in both cases; in one case, an older process. Both cases showed gastro-enteritis. No other infective processes were present. Both showed marked features of the hypoplastic lymphatic constitution. In both cases there were extreme congestion and edema of the central nervous system, with reticulo-endothelial proliferation. In both cases, no evidence of a polyneuritis were present. Both cases showed the same pathologic changes in the skin in the form of hyperkeratosis, slight pigmentation, chronic erythema without edema, hypertrophy of the sweat glands and perivascular reticulo-endothelial proliferation. Both cases showed inanition in the serious atrophy of the adipose tissues.

"The essential pathologic changes in these two cases would appear to be: Extreme edema and slight meningeal irritation of the central nervous system; chronic erythema of the skin, with hyperkeratosis; hypertrophy of the epidermis and sweat glands, with slight pigmentation of the rete, occurring in children of the hypoplastic lymphatic constitution, with associated or terminal respiratory infections and gastro-intestinal catarrh and inanition.

"These two cases, therefore, do not support the theory of a polyneuritis as the essential feature of the disease; neither do they justify the use of the term erythredema as a fitting designation, as they showed no edema of the corium. They do not present any evidence as

to a specific infectious etiology; they likewise show no relationship between tonsillar disease and the syndrome.

"The changes in the skin and central nervous system suggested to me at once the pathology of the early erythema stage of pellagra. The changes in the skin are also identical with those seen in certain forms of light sensitization, xeroderma and fagopyrism. They are also identical with certain stages of roentgen-ray and ultraviolet-ray erythemas. The entire anatomic picture in these two cases suggests either a food deficiency or a toxic state acting on persons of the hypoplastic constitution, affecting the reticulo-endothelial system of meninges and the skin, the vegetative nervous system and possibly leading to a light sensitization. May it not be, as was early suggested by Byfield, a condition closely related to pellagra, perhaps an infantile variety of that affection? Other necropsy cases must be collected and studied before this question can be settled. In the meantime, it would be better to call the condition Swift's disease than to designate it by terms that apply so little to it as do 'acrodynia' or 'erythredema.'"

It is because of these two cases, and the autopsy findings, which may be said to be the first to furnish any data at all reliable, that I have described the disease as a disturbance of nutrition in this volume. It remains for the future to determine whether this has been justifiable.

**Diagnosis.**—This should offer no particular difficulties to any one who has seen a well-marked case, as the picture once seen is not easily forgotten. Since the whole matter is far from settled and doubt still exists, it is only fair to present the points which are supposed to differentiate the condition from pellagra and so I reproduce this differentiation, as furnished by Weston (Abt's Pediatrics, Vol. II, p. 991):

<i>Acrodynia</i>	<i>Pellagra</i>
Occurs at any season.	Usually commences in spring or autumn.
No insanity.	Insanity often present.
No tendency to recurrence.	Recurrence usual.
Rash most pronounced at ends of fingers, gradually fading until it disappears above wrists.	Symmetrical rash with sharp line of demarcation on forearms and legs. No tendency to fade.
Dorsal surface of feet and ankles seldom involved.	Dorsal surface of feet and ankles usually involved.

*Acrodynia*
*Pellagra*

Erythematous rash may be present over entire body, with the exception of back of neck.	Not observed in pellagra.
Palms and soles the seat of intense burning, itching or perhaps numbness.	Not present in pellagra.
Diarrhea not usually serious.	Obstinate diarrhea often present.
Uncomplicated cases invariably recover.	Mortality comparatively high.
Photophobia present in two-thirds of cases.	Photophobia usually absent.
Great wretchedness in nearly all cases.	Wretchedness seldom occurs.
Absence of appetite the rule.	Not usually impaired in children.

By reference to description of pellagra it will be noted that some of these points are not striking.

**Treatment.**—The pain is apparently the result of the skin lesions and the parathesias, and so the treatment of these manifestations accomplishes more than other treatment. Cold calamine lotion is recommended by those who have had the most experience with the disease. The soothing effect of the application brings comfort, and permits the patient to sleep. The usual sedatives are not recommended. Since anorexia is a serious symptom and the nutrition is interfered with to an alarming extent, the feeding problem is the one to be solved ahead of any other. "Thick cereals, fruit juices and substances containing the fat soluble and water soluble vitamins should be offered." A balanced diet is, therefore, apparently to be sought and administered. The child's nutrition must be maintained, and resort to the stomach tube should not be postponed too long.

In the light of Goldberger's recent work in demonstrating the P—P factor and its influence on pellagra, it would appear to be wise to try a solution of yeast in the very young, and fresh lean meat, or at least the expressed juices from meat, perhaps combined with yeast, in older children. This is merely a suggestion in view of the autopsy findings in Warthin's two cases, and the pathological resemblance to the early stage of pellagra.





## APPENDIX

### VITAMIN CONTENT OF VARIOUS FOODS

BULLETIN OF THE UNITED STATES DEPARTMENT OF AGRICULTURE

BY

D. BREESE JONES, CHEMIST.

Significance of the symbols used in the table:

- Indicates no appreciable amount of vitamin.
- + Indicates that the vitamin is present in small amounts.
- ++ Indicates that the food is a fair source of vitamin.
- +++ Indicates that the vitamin is present in relatively large amount.
- ++++ Indicates that the vitamin is present in abundance.
- +++++ Indicates an exceptionally large amount of the vitamin.

The absence of any symbol indicates lack of available information.

SOURCE	VITAMIN A	VITAMIN B	VITAMIN C
<i>Vegetables</i>			
Spinach .....	+++++	+++	+++++
Lettuce .....	+++++	+++	+++++
Cabbage .....	+++++	+++++	+++++
Potatoes, white .....	+	+++	+++
Beans, kidney .....		+++	
Beans, navy .....		+++	—
Beans, soy .....	+++	+++	—
Beans, string .....	++	++	++
Beans, sprouted .....			++
Artichokes .....	++	+	
Beets .....	—	+	—
Carrots .....	+++++	+++++	++
Dandelion .....	+++	+	+
Onions .....		+	+++
Peas, fresh .....	+++++	+++	+++
Asparagus .....		+++++	
Parsley .....		+++++	
Parsnips .....	++	+++	
Potatoes, sweet .....	+++++	++	++
Celery .....		++	
Swedes .....		++	+++++
Squash, Hubbard .....	++		

SOURCE	VITAMIN A	VITAMIN B	VITAMIN C
<i>Vegetables—Continued</i>			
Egg plant (dried) .....		++	
Cauliflower .....	+	++	+
Chard .....	++	+	
Cucumber .....		+	
Endive .....	+		+
Radish .....	+ ?	+++	
Rutabaga .....	+	++	++++
Lentils .....	+++	+++	
Mushrooms .....	—	+++	
<i>Fats and Oils</i>			
Cod liver oil .....	+++++	—	—
Butter .....	+++++	—	—
Coconut oil .....	—	—	—
Corn oil .....	+	—	—
Cottonseed oil .....	+ ?	—	—
Lard .....	+ ?	—	—
Margarine, nut .....	—	—	—
Margarine, oleo .....	+	—	—
Orange peel oil .....	++		
Mutton fat .....	++	—	—
Beef fat .....	++	—	—
<i>Fruits</i>			
Apples .....	+	++	++
Cloudberries .....			++++
Bananas .....	+	+	++
Grapefruit .....		+++	+++++
Grape juice .....		++	+
Lemon juice .....		+++	+++++
Pineapples .....	++	++	+++
Cherries, fresh and canned .....			+
Limes .....		++	+
Orange juice .....	++	+++	+++++
Pears .....		++	++
Prunes .....		+	
Raspberries .....			++++
Strawberries .....			+++
Peaches .....			+++
Loganberries .....			+
Tomatoes, raw .....	+++++	+++	+++++
Tomatoes, canned .....	+++++	+++	+++++
Raisins .....	—	++	+
<i>Grains and Seeds</i>			
Wheat, whole .....	+	++++	—
Wheat, germ .....	++	+++++	—
Wheat, bran .....	++	+++	—
Corn, white .....	—	++++	—
Corn, yellow .....	++	++++	—
Oats .....	o to +	+++	—
Barley .....	+	+++	—
Rice, whole .....	+	+++	—
Rice, polished .....	—	—	—
Rice, bran .....	—	+++++	—
Millet seed .....	++	—	—

SOURCE	VITAMIN A	VITAMIN B	VITAMIN C
<i>Grains and Seeds—Continued</i>			
Cottonseed .....	+	+++	
Sunflower seed .....	+		
Kaffir .....		+++	
Rye .....	+	++	—
<i>Meat, fish, etc.</i>			
Lean muscle .....	+	+	+ ?
Pork, lean .....	+	+++	+
Pork, fat .....	++		
Chicken .....		—	
Turkey .....		—	
Duck .....		—	
Brains .....	++	+++	+ ?
Liver .....	+++++	+++++	+++
Heart .....	++++	++++	+
Kidney .....	+++	+++	+
Fish, lean .....	—	+	—
Fish, roe .....	++++	++	
Oysters .....			+++
<i>Nuts</i>			
Almonds .....	+	+++	
Brazil nuts .....		+++	
Chestnuts .....		+++	
Coconuts .....	++	+++	
Filberts .....		+++	
Hickory nuts .....	+	+++	+
Peanuts .....	+	+++	
Pecans .....		+++	
Walnuts, English .....	—	+++	
Walnuts, black .....		+++	
Pine nuts .....	+	+	+
<i>Dairy Products</i>			
Milk, whole .....	+++	+++	+
Milk, skim .....	+	+++	+
Milk, condensed, sweetened .....	+++	+++	+
Milk, evaporated .....	+++	+++	
Milk, powdered .....	+++	+++	+ ?
Butter .....	+++++	—	—
Cheese, whole milk .....	+++	+	—
<i>Miscellaneous</i>			
Yeast .....	—	+++++	—
Molasses, cane (blackstrap) .....		++++	
Molasses, beet .....		+++	
Molasses, sorghum .....		+	
Eggs .....	++++	++	—
Eggs, yolk .....	+++++	+++	—
Alfalfa .....	+++++	+++++	++++
Clover .....	+++++	++++	++++
Green grass .....	+++++	++++	++++
Tankage .....	—	—	
Blood meal .....	—	—	—
Timothy .....	+++++	++++	+++
Honey .....	—	+ ?	—

THE CHEMICAL COMPOSITION OF FOOD MATERIALS <sup>1</sup>

FOODS	PROTEIN	FAT	CARBO- HYDRATE	FUEL VALUE (CALORIES) PER POUND
<b>Animal foods</b>				
Tenderloin of beef, fresh .....	16.2	24.4		1,330
Tongue .....	18.9	9.2		740
Roast beef, cooked .....	22.3	28.6		1,620
Round steak, cooked .....	27.6	7.7		840
Sirloin steak .....	23.5	20.4		1,300
Dried beef, canned .....	39.2	5.4		960
Corned beef .....	15.6	26.2		1,395
Roast veal .....	20.4	5.6		615
Chops, lamb, broiled .....	21.7	29.9		1,665
Leg, lamb, cooked .....	19.7	12.7		900
Mutton, cooked .....	25.0	22.6		1,420
Roast pork .....	18.9	13.0		900
Ham, smoked, boiled .....	20.2	22.4		1,320
Salt pork .....	8.4	67.1		2,985
Bacon, smoked .....	10.5	64.8		2,930
Sausage, pork and beef together ...	19.4	24.1		1,380
Chicken, broilers and roast .....	21.5	2.5		505
Fowls, roast .....	13.7	12.3		775
Turkey, roast .....	27.8	18.4		1,295
Cod fish, fresh .....	16.7	.3		325
Haddock .....	8.4	.2		165
Halibut, steaks .....	15.3	4.4		470
Mackerel .....	11.6	3.5		365
Shad .....	9.4	4.8		380
Bluefish, cooked .....	25.9	4.5		670
Salmon, canned .....	21.8	12.1		915
Sardines, canned .....	23.0	19.7		1,260
Clams, in shell, fresh .....	8.6	1.0		240
Oysters, fresh .....	6.0	1.3		230
Eggs, hen's, fresh .....	13.4	10.5		720
<b>Dairy products</b>				
Butter .....	1.0	85.0		3,605
Buttermilk .....	3.0	.5	4.8	165
Cheese, American .....	28.8	35.9	.3	2,055
Cheese, cottage .....	20.9	1.0	1.8	510
Cheese, cream .....	18.7	27.4	2.4	1,530
Cream .....	2.5	18.5	4.5	910
Ice cream .....	9.0	83.0	11.7	150
Koumiss .....	2.8	2.1	5.4	240
Milk .....	8.8	8.3	54.1	1,520
Milk, skimmed .....	3.4	.3	5.1	170
Milk, whole .....	3.3	4.0	5.0	325
Whey .....	1.0	.3	5.0	125
<b>Miscellaneous</b>				
Gelatin .....	91.4	.1		1,705
Lard, refined .....		100.0		4,220
Beef juice .....	4.9	.6		115

<sup>1</sup> Some of these analyses are based on an average between the maximum and minimum contents, others obviously refer to edible portion only. Adopted from U. S. Bulletin (Atwater) Department of Agriculture.



FOODS	PROTEIN	FAT	CARBO- HYDRATE	FUEL VALUE (CALORIES) PER POUND
<b>Vegetable foods</b>				
Corn flour .....	7.1	1.3	78.4	1,645
Corn meal .....	9.2	1.9	75.4	1,655
Corn flakes .....	9.6	1.1	78.3	1,680
Hominy, cooked .....	2.2	.2	17.8	380
Oatmeal .....	16.1	7.2	67.5	1,860
Oatmeal, boiled .....	2.8	.5	11.5	285
Oatmeal gruel .....	1.2	.4	6.3	155
Miscellaneous, oats .....	16.3	7.3	66.8	1,855
Rice, boiled .....	2.8	.1	24.4	510
All analyses, wheat flour .....	10.8	1.1	74.8	1,640
Farina .....	11.0	1.4	76.3	1,685
Shredded wheat .....	10.5	1.4	77.9	1,700
Macaroni .....	13.4	.9	74.1	1,665
Macaroni, cooked .....	3.0	1.5	15.8	415
<i>Bread, crackers, pastry, etc.</i>				
Bread, brown .....	5.4	1.8	47.1	1,050
Bread, corn, johnnycake .....	7.9	4.7	46.3	1,205
Bread, rye .....	9.0	.6	53.2	1,180
Gluten bread .....	9.3	1.4	49.8	1,160
Graham bread .....	8.9	1.8	52.1	1,210
Rolls, Vienna .....	8.5	2.2	56.5	1,300
White bread, biscuit .....	8.0	1.4	54.3	1,220
White bread, miscellaneous .....	9.3	1.2	52.7	1,205
Whole wheat bread .....	9.7	.9	49.7	1,140
Zwieback .....	9.8	9.9	73.5	1,970
Boston crackers .....	11.0	8.5	71.1	1,885
Graham crackers .....	10.0	9.4	73.8	1,955
Oatmeal crackers .....	11.8	11.1	69.0	1,970
Pilot bread .....	11.1	5.0	74.2	1,800
Saltines .....	10.6	12.7	68.5	2,005
Soda crackers .....	9.8	9.1	73.1	1,925
Chocolate layer cake .....	6.2	8.1	64.1	1,650
Drop cake .....	5.9	9.0	64.8	1,695
Gingerbread .....	5.8	9.0	63.5	1,670
Sponge cake .....	6.3	10.7	65.9	1,795
Miscellaneous cookies .....	6.7	9.6	72.4	1,875
Lady fingers .....	8.8	5.0	70.6	1,685
Macaroons .....	6.5	15.2	65.2	1,975
Doughnuts .....	6.7	21.0	53.1	2,000
Pie, apple .....	3.1	9.8	42.8	1,270
Pie, cream .....	4.4	11.4	51.2	1,515
Pie, custard .....	4.2	6.3	26.1	830
Pie, lemon .....	3.6	10.1	37.4	1,190
Pie, mince .....	5.8	12.3	38.1	1,335
Pie, squash .....	4.4	8.4	21.7	840
Pudding, Indian meal .....	5.5	4.8	27.5	815
Pudding, rice custard .....	4.0	4.6	31.4	825
Pudding, tapioca .....	3.3	3.2	28.2	720
Pudding, tapioca, with apples .....	.3	.1	29.3	555
<i>Sugars and starches</i>				
Honey .....	.4		81.2	1,520

THE CHEMICAL COMPOSITION OF FOOD MATERIALS  
(Continued)

FOODS	PROTEIN	FAT	CARBO- HYDRATE	FUEL VALUE (CALORIES) PER POUND
<i>Sugars and starches—Con.</i>				
Starch, cornstarch .....			90.0	1,675
Sugar, coffee or brown .....			95.0	1,765
Sugar, granulated .....			100.0	1,860
Sugar, maple .....			82.8	1,540
Sugar, powdered .....			100.0	1,860
Sirup, maple .....			71.4	1,330
<i>Vegetables</i>				
Artichokes .....	2.6	.2	16.7	365
Asparagus, fresh .....	1.8	.2	3.3	105
Beans, butter, green .....	9.4	.6	29.1	740
Beans, dried .....	22.5	1.8	59.6	1,605
Beans, lima, dried .....	18.1	1.5	65.9	1,625
Beans, lima, fresh .....	7.1	.7	22.0	570
Beans, string, fresh .....	2.3	.3	7.4	195
Beets, cooked .....	2.3	.1	7.4	185
Beets, fresh .....	1.6	.1	9.7	215
Carrots, fresh .....	1.1	.4	9.3	210
Cauliflower .....	1.8	.5	4.7	140
Celery .....	1.1	.1	3.3	85
Corn, green .....	3.1	1.1	19.7	470
Cucumbers .....	.8	.2	3.1	80
Greens, beet, cooked .....	2.2	3.4	3.2	245
Greens, dandelion .....	2.4	1.0	10.6	285
Greens, turnip-salad .....	4.2	.6	6.3	220
Lettuce .....	1.2	.3	2.9	90
Mushrooms .....	3.5	.4	6.8	210
Onions, fresh .....	1.6	.3	9.9	225
Onions, green, New Mexico .....	1.0	.1	11.2	230
Parsnips .....	1.6	.5	13.5	300
Peas, dried .....	24.6	1.0	62.0	1,655
Peas, green .....	7.0	.5	16.9	465
Potatoes, raw or fresh .....	2.2	.1	18.4	385
Potatoes, cooked, boiled .....	2.5	.1	20.9	440
Potatoes, sweet, cooked and prepared	3.0	2.1	42.1	925
Rhubarb .....	.6	.7	3.6	105
Spinach, cooked .....	2.1	4.1	2.6	260
Squash .....	1.4	.5	9.0	215
Tomatoes, fresh .....	.9	.4	3.9	105
Turnips .....	1.3	.2	8.1	185
Asparagus, canned .....	1.5	.1	2.8	85
Beans, baked, canned .....	6.9	2.5	19.6	600
Beans, string, canned .....	1.1	.1	3.8	95
Beans, lima, canned .....	4.0	.3	14.6	360
Corn, green, canned .....	2.8	1.2	19.0	455
Peas, green, canned .....	3.6	.2	9.8	255
Squash, canned .....	.9	.5	10.5	235
Tomatoes, canned .....	.2	.2	4.0	105
Cabbage .....	1.6	.3	5.6	145
Olives, green .....	.8	20.2	8.5	1,025

FOODS	PROTEIN	FAT	CARBO- HYDRATE	FUEL VALUE (CALORIES) PER POUND
<i>Fruits, berries, etc.</i> .....				
Apples, fresh .....	.4	.5	14.2	290
Apples, as purchased .....	.3	.3	10.8	220
Apricots, fresh .....	1.1		13.4	270
Bananas, yellow .....	1.3	.6	22.0	460
Blackberries, fresh .....	1.3	1.0	10.9	270
Cherries, fresh .....	1.0	.8	16.7	365
Cranberries, fresh .....	.4	.6	9.9	215
Grapes, fresh .....	1.0	1.2	14.4	335
Lemons .....	1.0	.7	8.5	205
Lemon juice .....			9.8	180
Muskmelons, as purchased .....	.3		4.6	90
Oranges, average .....	.8	.2	11.6	240
Oranges, as purchased .....	.6	.1	8.5	170
Peaches, average .....	.7	.1	9.4	190
Peaches, as purchased .....	.5	.1	7.7	155
Pears .....	.6	.5	14.1	295
Pineapple .....	.4	.3	9.7	200
Raspberries, red .....	1.0		12.6	255
Raspberries, black .....	1.7	1.0	12.6	310
Strawberries .....	1.0	.6	7.4	180
Watermelons .....	.4	.2	6.7	140
Apples, dried .....	1.6	2.2	66.1	1,350
Apricots, dried .....	4.7	1.0	62.5	1,290
Dates, dried .....	2.1	2.8	78.4	1,615
Figs, dried .....	4.3	.3	74.2	1,475
Prunes, dried .....	1.8		62.2	1,190
Raisins, dried .....	2.3	3.0	68.5	1,445
Blackberries, canned .....	.8	2.1	56.4	1,150
Blueberries .....	.6	.6	12.8	275
Cherries .....	1.1	.1	21.1	415
Jelly, first quality .....	1.1		77.2	1,455
Marmalade (orange peel) .....	.6	.1	84.5	1,585
Peaches .....	.7	.1	10.8	220
Pears .....	.3	.3	18.0	355
Pineapples .....	.4	.7	36.4	715
Strawberries, stewed .....	.7		24.0	460
<i>Nuts</i> .....				
Almonds .....	21.0	54.9	17.3	3,030
Brazil nuts .....	17.0	66.8	7.0	3,265
Filberts .....	15.6	65.3	13.0	3,290
Peanuts .....	25.8	38.6	24.4	2,560
Peanut butter .....	29.3	46.5	17.1	2,825
Pecans, unpolished .....	9.6	70.5	15.3	3,435
Walnuts, California .....	18.4	64.4	13.0	3,300
<i>Miscellaneous</i> .....				
Chocolate .....	12.9	48.7	30.3	2,860
Cocoa .....	21.6	28.9	37.7	2,320
Infants and invalids' foods .....	12.7	3.3	76.2	1,795

## EQUIVALENT TABLE IN CENTIMETERS AND INCHES

Cm.	In.	Cm.	In.	Cm.	In.
0		0			
1	$\frac{1}{2}$	16		31	
2	$\frac{3}{4}$	17		32	
3	1	18	7	33	13
4		19		34	
5	2	20	8	35	
6		21		36	14
7		22		37	
8	3	23	9	38	15
9		24		39	
10	4	25	10	40	
11		26		41	16
12		27		42	
13	5	28	11	43	17
14		29		44	
15	6	30	12	45	18



Cm.	In.	Cm.	In.	Cm.	In.
46					
47		62		77	
48	19	63	25	78	
49		64		79	31
50		65		80	
51	20	66	26	81	32
52		67		82	
53	21	68	27	83	
54		69		84	33
55		70		85	
56	22	71	28	86	34
57		72		87	
58	23	73	29	88	
59		74		89	35
60		75		90	
61	24	76	30	91	36

## CONVERSION TABLE: AVOIRDUPOIS TO METRIC WEIGHTS

Pounds	Ounces	Kilograms	Pounds	Ounces	Kilograms
0	+				
	$\frac{1}{2}$	== .014		$13\frac{1}{2}$	== .826
1		== .028		14	== .840
	$1\frac{1}{2}$	== .042		$14\frac{1}{2}$	== .854
	2	== .056		15	== .868
	$2\frac{1}{2}$	== .070		$15\frac{1}{2}$	== .882
	3	== .084	2	0	== .907
	$3\frac{1}{2}$	== .098		$\frac{1}{2}$	== .921
	4	== .112		1	== .935
	$4\frac{1}{2}$	== .126		$1\frac{1}{2}$	== .949
	5	== .140		2	== .963
	$5\frac{1}{2}$	== .154		$2\frac{1}{2}$	== .978
	6	== .168		3	== .992
	$6\frac{1}{2}$	== .182		$3\frac{1}{2}$	== 1.007
	7	== .196		4	== 1.021
	$7\frac{1}{2}$	== .210		$4\frac{1}{2}$	== 1.035
	8	== .224		5	== 1.049
	$8\frac{1}{2}$	== .238		$5\frac{1}{2}$	== 1.063
	9	== .252		6	== 1.077
	$9\frac{1}{2}$	== .266		$6\frac{1}{2}$	== 1.091
10		== .280		7	== 1.105
$10\frac{1}{2}$		== .294		$7\frac{1}{2}$	== 1.119
11		== .308		8	== 1.133
$11\frac{1}{2}$		== .322		$8\frac{1}{2}$	== 1.148
12		== .336		9	== 1.162
$12\frac{1}{2}$		== .350		$9\frac{1}{2}$	== 1.176
13		== .364	10		== 1.190
$13\frac{1}{2}$		== .378		$10\frac{1}{2}$	== 1.204
14		== .392		11	== 1.219
$14\frac{1}{2}$		== .406		$11\frac{1}{2}$	== 1.233
15		== .420		12	== 1.247
$15\frac{1}{2}$		== .434		$12\frac{1}{2}$	== 1.261
1	+			13	== 1.275
	$\frac{1}{2}$	== .448		$13\frac{1}{2}$	== 1.289
	1	== .462		14	== 1.304
	$1\frac{1}{2}$	== .476		$14\frac{1}{2}$	== 1.318
	2	== .490		15	== 1.332
	$2\frac{1}{2}$	== .504		$15\frac{1}{2}$	== 1.346
	3	== .518	3	+	
	$3\frac{1}{2}$	== .532		0	== 1.360
	4	== .546		$\frac{1}{2}$	== 1.374
	$4\frac{1}{2}$	== .560		1	== 1.389
	5	== .574		$1\frac{1}{2}$	== 1.403
	$5\frac{1}{2}$	== .588		2	== 1.417
	6	== .602		$2\frac{1}{2}$	== 1.431
	$6\frac{1}{2}$	== .616		3	== 1.445
	7	== .630		$3\frac{1}{2}$	== 1.459
	$7\frac{1}{2}$	== .644		4	== 1.474
	8	== .658		$4\frac{1}{2}$	== 1.488
	$8\frac{1}{2}$	== .672		5	== 1.502
	9	== .686		$5\frac{1}{2}$	== 1.516
	$9\frac{1}{2}$	== .700		6	== 1.530
10		== .714		$6\frac{1}{2}$	== 1.545
$10\frac{1}{2}$		== .728		7	== 1.559
11		== .742		$7\frac{1}{2}$	== 1.573
$11\frac{1}{2}$		== .756		8	== 1.587
12		== .770		$8\frac{1}{2}$	== 1.601
$12\frac{1}{2}$		== .784		9	== 1.615
13		== .798		$9\frac{1}{2}$	== 1.630
		== .812			

Pounds	Ounces	Kilograms	Pounds	Ounces	Kilograms
	10	= 1.64425		8	= 2.494
	10½	= 1.65843		8½	= 2.508
	11	= 1.67260		9	= 2.523
	11½	= 1.68678		9½	= 2.537
	12	= 1.70095		10	= 2.551
	12½	= 1.71513		10½	= 2.565
	13	= 1.72930		11	= 2.579
	13½	= 1.74347		11½	= 2.593
	14	= 1.75765		12	= 2.608
	14½	= 1.77182		12½	= 2.622
	15	= 1.78600		13	= 2.636
	15½	= 1.80017		13½	= 2.650
4 +	0	= 1.81437		14	= 2.664
	½	= 1.82852		14½	= 2.679
	1	= 1.84269		15	= 2.693
	1½	= 1.85687		15½	= 2.707
	2	= 1.87105	6 +	0	= 2.721
	2½	= 1.885		½	= 2.735
	3	= 1.899		1	= 2.749
	3½	= 1.913		1½	= 2.764
	4	= 1.927		2	= 2.778
	4½	= 1.941		2½	= 2.792
	5	= 1.956		3	= 2.806
	5½	= 1.970		3½	= 2.820
	6	= 1.984		4	= 2.834
	6½	= 1.998		4½	= 2.849
	7	= 2.012		5	= 2.863
	7½	= 2.026		5½	= 2.877
	8	= 2.041		6	= 2.891
	8½	= 2.055		6½	= 2.905
	9	= 2.069		7	= 2.919
	9½	= 2.083		7½	= 2.934
	10	= 2.097		8	= 2.948
	10½	= 2.112		8½	= 2.962
	11	= 2.126		9	= 2.976
	11½	= 2.140		9½	= 2.990
	12	= 2.154		10	= 3.005
	12½	= 2.168		10½	= 3.019
	13	= 2.182		11	= 3.033
	13½	= 2.197		11½	= 3.047
	14	= 2.211		12	= 3.061
	14½	= 2.225		12½	= 3.075
	15	= 2.239		13	= 3.090
	15½	= 2.253		13½	= 3.104
5 +	0	= 2.267		14	= 3.118
	½	= 2.282		14½	= 3.132
	1	= 2.296		15	= 3.146
	1½	= 2.310		15½	= 3.160
	2	= 2.324	7 +	0	= 3.175
	2½	= 2.338		½	= 3.189
	3	= 2.353		1	= 3.203
	3½	= 2.367		1½	= 3.217
	4	= 2.381		2	= 3.231
	4½	= 2.395		2½	= 3.246
	5	= 2.409		3	= 3.260
	5½	= 2.423		3½	= 3.274
	6	= 2.438		4	= 3.288
	6½	= 2.452		4½	= 3.302
	7	= 2.466		5	= 3.316
	7½	= 2.480		5½	= 3.331

Pounds	Ounces	Kilograms	Pounds	Ounces	Kilograms
	6	= 3.345		4	= 4.195
	6½	= 3.358		4½	= 4.209
	7	= 3.373		5	= 4.224
	7½	= 3.387		5½	= 4.238
	8	= 3.401		6	= 4.252
	8½	= 3.416		6½	= 4.266
	9	= 3.430		7	= 4.280
	9½	= 3.444		7½	= 4.294
	10	= 3.458		8	= 4.309
	10½	= 3.472		8½	= 4.323
	11	= 3.486		9	= 4.337
	11½	= 3.501		9½	= 4.351
	12	= 3.515		10	= 4.365
	12½	= 3.529		10½	= 4.379
	13	= 3.543		11	= 4.394
	13½	= 3.557		11½	= 4.408
	14	= 3.572		12	= 4.422
	14½	= 3.586		12½	= 4.436
	15	= 3.600		13	= 4.450
	15½	= 3.614		13½	= 4.465
				14	= 4.479
8 +	0	= 3.628		14½	= 4.493
	½	= 3.642		15	= 4.507
	1	= 3.657		15½	= 4.521
	1½	= 3.671			
	2	= 3.685	10 +	0	= 4.535
	2½	= 3.699		½	= 4.549
	3	= 3.713		1	= 4.563
	3½	= 3.727		1½	= 4.577
	4	= 3.742		2	= 4.592
	4½	= 3.756		2½	= 4.606
	5	= 3.770		3	= 4.620
	5½	= 3.784		3½	= 4.634
	6	= 3.798		4	= 4.648
	6½	= 3.813		4½	= 4.662
	7	= 3.827		5	= 4.677
	7½	= 3.841		5½	= 4.691
	8	= 3.855		6	= 4.705
	8½	= 3.869		6½	= 4.719
	9	= 3.883		7	= 4.733
	9½	= 3.898		7½	= 4.747
	10	= 3.912		8	= 4.762
	10½	= 3.926		8½	= 4.776
	11	= 3.940		9	= 4.790
	11½	= 3.954		9½	= 4.804
	12	= 3.968		10	= 4.818
	12½	= 3.983		10½	= 4.833
	13	= 3.997		11	= 4.847
	13½	= 4.011		11½	= 4.861
	14	= 4.025		12	= 4.875
	14½	= 4.039		12½	= 4.889
	15	= 4.053		13	= 4.904
	15½	= 4.068		13½	= 4.920
				14	= 4.934
9 +	0	= 4.082		14½	= 4.948
	½	= 4.096		15	= 4.963
	1	= 4.110		15½	= 4.977
	1½	= 4.124			
	2	= 4.139	11 +	0	= 4.991
	2½	= 4.153		½	= 5.006
	3	= 4.167		1	= 5.020
	3½	= 4.181		1½	= 5.034



Pounds	Ounces	Kilograms	Pounds	Ounces	Kilograms
	2	= 5.047	13	+	0 = 5.883
	2½	= 5.061		4	= 5.996
	3	= 5.075		8	= 6.108
	3½	= 5.089		12	= 6.221
	4	= 5.103	14	+	0 = 6.333
	4½	= 5.117		4	= 6.446
	5	= 5.132		8	= 6.558
	5½	= 5.146		12	= 6.671
	6	= 5.160	15	+	0 = 6.783
	6½	= 5.174		4	= 6.896
	7	= 5.188		8	= 7.009
	7½	= 5.202		12	= 7.121
	8	= 5.216	16	+	0 = 7.233
	8½	= 5.230		4	= 7.346
	9	= 5.244		8	= 7.458
	9½	= 5.258		12	= 7.571
	10	= 5.273	17	+	0 = 7.683
	10½	= 5.287		4	= 7.795
	11	= 5.301		8	= 7.908
	11½	= 5.315		12	= 8.020
	12	= 5.329	18	+	0 = 8.133
	12½	= 5.343		4	= 8.245
	13	= 5.357		8	= 8.357
	13½	= 5.371		12	= 8.470
	14	= 5.385	19	+	0 = 8.582
	14½	= 5.399		4	= 8.694
	15	= 5.414		8	= 8.807
	15½	= 5.428		12	= 8.919
12	+	0 = 5.442	20	+	0 = 9.032
	½	= 5.456		4	= 9.144
	1	= 5.470		8	= 9.257
	1½	= 5.484		12	= 9.369
	2	= 5.498	21	+	0 = 9.482
	2½	= 5.512		4	= 9.594
	3	= 5.526		8	= 9.707
	3½	= 5.540		12	= 9.819
	4	= 5.554	22	+	0 = 9.932
	4½	= 5.568		4	= 10.044
	5	= 5.583		8	= 10.157
	5½	= 5.597		12	= 10.269
	6	= 5.601	23	+	0 = 10.382
	6½	= 5.615		4	= 10.494
	7	= 5.629		8	= 10.607
	7½	= 5.643		12	= 10.719
	8	= 5.657	24	+	0 = 10.832
	8½	= 5.671		4	= 10.944
	9	= 5.685		8	= 11.057
	9½	= 5.699		12	= 11.169
	10	= 5.714			
	10½	= 5.728			
	11	= 5.742			
	11½	= 5.756			
	12	= 5.770			
	12½	= 5.784			
	13	= 5.798			
	13½	= 5.812			
	14	= 5.826			
	14½	= 5.840			
	15	= 5.855			
	15½	= 5.869			

Pounds	Ounces	Kilograms	Pounds	Ounces	Kilograms
25	+	0 = 11.282	38	+	0 = 17.032
		4 = 11.395			4 = 17.145
		8 = 11.507			8 = 17.257
		12 = 11.620			12 = 17.370
26	+	0 = 11.732	39	+	0 = 17.482
		4 = 11.845			4 = 17.595
		8 = 11.957			8 = 17.707
		12 = 12.070			12 = 17.820
27	+	0 = 12.182	40	+	0 = 17.932
		4 = 12.295			4 = 18.045
		8 = 12.407			8 = 18.157
		12 = 12.520			12 = 18.270
28	+	0 = 12.632	41	+	0 = 18.382
		4 = 12.745			4 = 18.495
		8 = 12.857			8 = 18.607
		12 = 12.970			12 = 18.720
29	+	0 = 13.082	42	+	0 = 18.832
		4 = 13.195			4 = 18.945
		8 = 13.307			8 = 19.057
		12 = 13.420			12 = 19.170
30	+	0 = 13.532	43	+	0 = 19.282
		4 = 13.645			4 = 19.395
		8 = 13.757			8 = 19.507
		12 = 13.870			12 = 19.620
31	+	0 = 13.982	44	+	0 = 19.732
		4 = 14.095			4 = 19.845
		8 = 14.207			8 = 19.957
		12 = 14.320			12 = 20.070
32	+	0 = 14.432	45	+	0 = 20.182
		4 = 14.545			4 = 20.295
		8 = 14.657			8 = 20.407
		12 = 14.670			12 = 20.520
33	+	0 = 14.782	46	+	0 = 20.632
		4 = 14.895			4 = 20.745
		8 = 15.007			8 = 20.857
		12 = 15.120			12 = 20.970
34	+	0 = 15.232	47	+	0 = 21.082
		4 = 15.345			4 = 21.195
		8 = 15.457			8 = 21.307
		12 = 15.570			12 = 21.420
35	+	0 = 15.682	48	+	0 = 21.532
		4 = 15.795			4 = 21.645
		8 = 15.907			8 = 21.757
		12 = 16.020			12 = 21.870
36	+	0 = 16.132	49	+	0 = 21.982
		4 = 16.245			4 = 22.095
		8 = 16.357			8 = 22.207
		12 = 16.470			12 = 22.320
37	+	0 = 16.582	50	+	0 = 22.432
		4 = 16.695			
		8 = 16.807			
		12 = 16.920			

## EQUIVALENTS

Fluid Ounces		Cubic Centimeters	Fluid Ounces		Cubic Centimeters
$\frac{1}{2}$	=	15	18	=	540
1	=	30	$18\frac{1}{2}$	=	555
$1\frac{1}{2}$	=	45	19	=	570
2	=	60	$19\frac{1}{2}$	=	585
$2\frac{1}{2}$	=	75	20	=	600
3	=	90	$20\frac{1}{2}$	=	615
$3\frac{1}{2}$	=	105	21	=	630
4	=	120	$21\frac{1}{2}$	=	645
$4\frac{1}{2}$	=	135	22	=	660
5	=	150	$22\frac{1}{2}$	=	675
$5\frac{1}{2}$	=	165	23	=	690
6	=	180	$23\frac{1}{2}$	=	705
$6\frac{1}{2}$	=	195	24	=	720
7	=	210	$24\frac{1}{2}$	=	735
$7\frac{1}{2}$	=	225	25	=	750
8	=	240	$25\frac{1}{2}$	=	765
$8\frac{1}{2}$	=	255	26	=	780
9	=	270	$26\frac{1}{2}$	=	795
$9\frac{1}{2}$	=	285	27	=	810
10	=	300	$27\frac{1}{2}$	=	825
$10\frac{1}{2}$	=	315	28	=	840
11	=	330	$28\frac{1}{2}$	=	855
$11\frac{1}{2}$	=	345	29	=	870
12	=	360	$29\frac{1}{2}$	=	885
$12\frac{1}{2}$	=	375	30	=	900
13	=	390	$30\frac{1}{2}$	=	915
$13\frac{1}{2}$	=	405	31	=	930
14	=	420	$31\frac{1}{2}$	=	945
$14\frac{1}{2}$	=	435	32	=	960
15	=	450	$32\frac{1}{2}$	=	975
$15\frac{1}{2}$	=	465	33	=	990
16	=	480	$33\frac{1}{2}$	=	1005
$16\frac{1}{2}$	=	495	34	=	1020
17	=	510			
$17\frac{1}{2}$	=	525			





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